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BULLETIN OF
THE NEW YORK ACADEMY
OF MEDICINE

JUNE, 1942

NEUROCIRCULATORY ASTHENIA AND RELATED PROBLEMS IN MILITARY MEDICINE*

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INTRODUCTION

With the advent of the second World War the problems of neurocirculatory asthenia have returned in Europe, and have been revived in this country in connection with the examination of draftees and volunteers. It is a disorder of individual and social importance even in peace time; but with war or the threat of war, it becomes imperative for the Army and Navy to avoid cluttering up their combat services with men who may break down with neurocirculatory asthenia, become incapacitated, and finally through pensions and invalidism become a permanent financial burden to the country. These are practical reasons for again considering the subject, but in addition our concept of the condition has undergone modification since 1917 when Thomas Lewis[1,2] and others brought out the admirable reports of their experience with the effort syndrome in the British Army. Something more is now known of the etiology, pathologic physiology, symptomatology, signs,

* Read October 15, 1941 at the Graduate Fortnight of The New York Academy of Medicine.

diagnosis, prognosis, and possibly even of treatment than was known in 1917. These are the important reasons for reconsidering the whole subject at the present time.

Definition: Neurocirculatory asthenia is a clinical syndrome, not a disease sui generis, characterized by dyspnea, palpitation, chest pain, exhaustibility, dizziness, headache, nervousness, and a great variety of associated symptoms and signs called forth or increased by effort, and in which no evidence of structural disease can be found to which the symptoms may be related.

Terminology: A dozen other names have been used at various times by different authors. Out of deference to Da Costa one would be tempted to use the term he originally introduced, "The Irritable Heart," but there are great practical objections to the use of any name which directs attention to the heart. Very recently in his admirable lectures Dr. Paul Wood[3] has used the designation "Da Costa's Syndrome," partly to honor Da Costa, but also with the implication that the syndrome may possibly "become of historical interest only." There is in general a justifiable objection to the introduction of a patronymic name into the nomenclature if a more descriptive or specific one can be devised. Temporarily a patronymic name may be adopted, but only until such a time as the nature or etiology of the disease or disorder is clarified. Some of the other terms used to designate this condition are:

1. Effort Syndrome (Lewis)
2. Musculature Exhaustion of the Heart (Hartshorne)
3. X-Disease (Mackenzie)
4. Cardiovascular Neurosis (Caughey)
5. Neurasthenia Cordis
6. Orthostatic Tachycardia
7. Vasoneurosis (used especially for children)
8. Dilatative Weakness (used for adolescents)
9. The Soldier's Heart.
10. Autonomic Imbalance (Kessel and Hyman)
11. Die Herzneurose (of the Germant authors)
12. Disordered Action of the Heart ("D.A.H." of the British Army).

In 1918 we[4] suggested the term neurocirculatory asthenia and this was selected because it was descriptive, non-committal as regards the heart, and shortened to the cryptic "N.C.A." would be understood and

not misunderstood all along the line. It may be preferred to the effort syndrome with its possible implication that the symptomatology is that of an exaggeration of the normal response to physical effort. The very varied clinical picture presented by neurocirculatory asthenia probably represents something fundamentally different than the enhanced physiologic reactions to physical exertion, although there are many points of superficial resemblance. Neurocirculatory asthenia has been adopted by the U. S. Army and also for the forthcoming edition of the Standard Classified Nomenclature of Disease. It is included in brackets after "Effort Syndrome" in the Nomenclature of Diseases of the Heart published by the New York Heart Association.

Historical: This is not at all a new disorder. It is stated that even Hippocrates realized that palpitation might result from emotion, fright or excitement. The condition certainly occurs in its milder grades in civil life among adults and children, but becomes more intensified, better defined and exceedingly common under military conditions. Its prevalence in the Civil War was first recorded by Hartshorne[5] and by Da Costa[6] in 1864; the latter included it in the first edition of his textbook on "Medical Diagnosis" in 1864, but he waited until 1871 before he finally published his classic paper "On the Irritable Heart: a Clinical Study of a Form of Functional Cardiac Disorder and Its Consequences."[7] This still stands as a model of direct clinical observation. By following the subsequent histories of his cases over a period of years he collected some fifteen instances in which he thought the irritable heart was followed by cardiac hypertrophy. Da Costa cited this group to show that "disordered function leads to organic disease," i.e., to hypertrophy of the heart. He described the necropsy findings of one patient who died following an operation for strangulated hernia in which there was left ventricular hypertrophy. At the present time the evidence is against this view that neurocirculatory asthenia leads to hypertension and hypertrophy, although of course any individual with (or without) neurocirculatory asthenia may ultimately develop abnormally high blood pressure. Da Costa also included some doubtful or mixed cases—that is patients in whom the symptoms of the irritable heart were superimposed upon those of an organic cardiac condition. He realized that one may have the combination of an irritable heart and valvular disease of the heart. He also took up the differential diagnosis of the irritable heart and pulmonary tuberculosis, anemia and malingering. Hartshorne

(1864) excluded anemia or scurvy (both of which were prevalent among the soldiers of the Army of the Potomac) as etiologic factors in neurocirculatory asthenia, as the heart affection lasted long after the patients were cured of the anemia or scurvy. As a result of his observations on these soldiers Hartshorne says: "Judging from what I have seen, I think it can hardly be a mistaken opinion that they are entirely unfit for ordinary field service in the Army. No doubt many, perhaps most of them, would be quite able to do light service in various ways." Those conclusions of 1864 hold today, although one cannot accept Hartshorne's theory, that the condition is due to a "muscular exhaustion of the heart."

Da Costa found some reference to the condition in the British Blue Book of the Crimean War, and the disorder is mentioned by MacLean[8] in 1867, by Myers[9] in his volume "On the Etiology and Prevalence of Diseases of the Heart among Soldiers" (1870), by Tyson[10] during the South African War, and by Captain McCarthy[11] in soldiers returned from a campaign on the Indian frontier in 1898. These authors were apt to lay great stress on the accoutrements and tight tunics, and even the busby as a cause of the condition. Its prevalence in a severe form in the British, French and German armies during the first World War was accounted for by the conditions of modern warfare at that time—prolonged marching, trench life, intensive fighting, terrific bombardment, gassing, burial, and trench fever, dysentery and other infections.

What is neurocirculatory asthenia? It seems to me that an abnormal reactivity of the nervous system, including the innervation of the entire circulatory system may account for the varying neuro-psychosomatic symptoms. The symptoms are physiological and psychological.[12] This irritability or instability of the nervous system may originate in an increased stimulation of the cerebral cortex. From the cortex the stimuli may pass to the hypothalamus which is a center for the autonomic nervous system. The hypothalamus is connected on the one hand with the cortex and on the other hand probably with the vagal nuclei and the sympathetic. So, disturbances in the hypothalamic region may affect respiration, pilomotor reflexes, vasomotor reflexes, sudomotor and other reflexes. In this way one may explain to one's self the great variety of symptoms of neurocirculatory asthenia, some of them characteristic of vagotonia, some of sympathicotonia. That is one possible mechanism. Another possible mechanism is the humoral, but this seems less likely.

Further pharmacological studies with adrenergic and cholinergic drugs may help to elucidate the problem.

The thesis that there are situational factors that will produce neurocirculatory asthenia in the individual because of a basic attitude toward that situation is tenable. Given a set of disagreeable circumstances such as warfare, threat of war, or merely drafting, why do some individuals present these symptoms? Partly because some are constitutionally more susceptible as will be shown later, but in addition there are other factors, such as motive. Indeed one does not have to resort to deep psychoanalytical investigations to uncover these latent conflicts. Just superficial consideration will detect emotional attitudes that can give rise to conflicts. If for example a man feels that he is enlisting in a cause for himself or his family, and that there is no alternative, or the alternative is even worse, then there is no conflict. If he is compelled to go to war against his own inclinations, then there may be a conflict and there is a basis for an anxiety state. Neurocirculatory asthenia seems to me to be the somatic component of a psychosomatic state. The more severe the neurocirculatory asthenia, the more it is an expression of visceral rather than of an articulate protest. Pre-war and war conditions make manifest symptoms referable to the cardiovascular system, just as they produce gastrointestinal symptoms, e.g., nausea, vomiting or diarrhea, genitourinary symptoms such as polyuria, and respiratory and sudomotor symptoms. There are undoubtedly many anxiety neuroses that have no somatic manifestations. Neurocirculatory asthenia is important because it ear-marks those men who are potential candidates for an anxiety neurosis or psychoneurosis. You may well ask, if neurocirculatory asthenia is merely a manifestation of a psychoneurotic state, why not turn the examination of registrants, draftees and recruits over to the neuropsychiatrists? It seems, however, preferable to leave the field of diagnosis in the hands of cardiologists and internists, as they are better equipped to rule out such organic diseases as early organic heart disease, pulmonary tuberculosis, the *formes frustes* of Graves' disease and anemia.

Family History and Previous Personal History; Psychoneurotic Factors: To corroborate the view that neurocirculatory asthenia is the somatic concomitant of anxiety and other psychoneuroses there is the evidence which the late Dr. Rothschild and I collected under Sir Thomas Lewis at Colchester in 1917. A comparison was made of the percentages of

TABLE I

PERCENTAGES OF CHARACTERISTICS NAMED IN FAMILY HISTORY OF PATIENTS SUFFERING FROM (1) NEUROSES, (2) "IRRITABLE HEART" OF SOLDIERS, AND (3) CONTROLS

	Neurosis Per Cent	*Irritable Heart Per Cent*	*Controls (Wounded) Per Cent*
Nervousness	64	45	15
Alcoholism (Parents and Grandparents)	50	15	24
Teetotalers (Parents and Grandparents)	30	15	16
Irritability of Temper	36	27	12
Insanity	34	23	0
Epilepsy	30	15	0
*Tuberculosis (Immediate Family)	12	13	4
*Tuberculosis (Relatives)	5	15	4
Stigmata	10	17	0
Positive History for One or Several of Foregoing	74	56	38

*Tuberculosis in the family history would probably not be included if such statistics were being collected today. Similarly nowadays the item "Married" would be omitted from Table II, although it is interesting to find the low percentage of married men among the neurocirculatory asthenics.

certain characteristics in the family history of soldiers suffering from (1) Psychoneurosis, (2) neurocirculatory asthenia or as it was called in 1917, the "irritable heart of soldiers," and (3) controls, i.e., soldiers invalided for war wounds. A positive family history of one or several of the characteristics selected was obtained in 74 per cent of the neuroses group (by Wolfsohn[13]), in 56 per cent of the neurocirculatory group, and in 38 per cent of the controls (see Table I). In the previous personal histories the contrast is much greater. There was a positive personal history in 76 per cent of psychoneuroses, 51 per cent of neurocirculatory asthenia, and only 12 per cent of the controls. A positive family *and* personal history was obtained in 70 per cent of the neurosis group, in 46 per cent of the neurocirculatory asthenias, but in only 6 per cent of the controls (See Table II). The practical military point was this: In such patients invalided for neurocirculatory asthenia as have neuropsychic factors in their family and previous personal histories, the

TABLE II

PERCENTAGES OF CHARACTERISTICS NAMED IN THE PERSONAL HISTORY IN CASES OF (1) NEUROSIS, OF (2) "IRRITABLE HEART" OF SOLDIERS AND OF (3) CONTROLS, I.E., WOUNDED

	Neurosis Per Cent	*Irritable Heart Per Cent*	*Controls (Wounded) Per Cent*
Stigmata	34	12	4
Previous Nervousness	66	46	12
Fears	50	31	8
Head Injury	38	5	12
Epilepsy and "Fits"	8	5	0
Tobacco (excessive)	8	1	4
Alcohol (excessive)	6	0	16
Alcohol (teetotaler)	48	36	20
Married	42	18	28
Moody	55	27	8
Previous Breakdown	2	21	0
Enuresis	12	14	4
Frights in Childhood	4	19	0
Excessive Religion	6	6	0
Positive Personal History	76	51	12
Positive Family and Personal History	70	46	6

value of the combatant service rendered in the past is found to be negligible. They are the group who in civilian life usually do sedentary or light work.[14]

Constitutional Asthenia: A history of constitutional physical asthenia was obtained in about 70 per cent of this group with psychoneurotic factors. By this rather vague term, "constitutional asthenia," is meant a relative inferiority or an anomaly in the assemblage of inherent characteristics, both functional and morphologic, which go to make up the organism. One may include in this term those who have always been short of breath, have been unable to play the more strenuous games, have fainted or become dizzy readily, have flushed easily, perspired too profusely and suffered from cold blue clammy hands. In addition one

may add those who have certain forms of "habitus," such as the splanchnoptotic, the feminine, the eunuchoid; the flat chested, reedy type, and those with drop hearts.

Juvenile Vasoneurotics: Bass and Wessler[15] in 1913 were the first American clinicians to draw attention to a group of children presenting the symptoms of neurocirculatory asthenia, who in addition showed orthostatic albuminuria. In 1918 we were able to show that a considerable percentage of soldiers invalided for neurocirculatory asthenia had shown symptoms of constitutional asthenia before the age of seventeen. In other words, many patients with neurocirculatory asthenia are the adult individuals who during their developmental period presented vasoneurotic symptoms. Some in this group had had symptoms as long as they could remember, but usually the symptoms of this constitutional weakness manifested themselves between the ages of eight and sixteen years. Since then we have seen many instances in civilian practice of children suffering from this condition, some of whom have continued to suffer into adult life. The natural history of this disorder from childhood to adult life and even old age requires further investigation and description.

Clinical Picture: A clinical description of this disorder as it occurs in its mild form among civilians, for example after influenza, is hardly necessary because it is familiar to general practitioners. It was a memorable picture, however, to see the severe and incapacitating type of the same disorder at Colchester among British soldiers, invalided from France in 1917-18. Many arrived on stretchers, completely done in physically and psychically. Their predominant symptoms could be referred to the circulatory and nervous systems. They complained of dyspnea, palpitation, chest pain, extreme fatigue, dizziness, nervousness and headaches; and this was the order of frequency of their chief symptoms. We were struck by the cold blue clammy hands, by the axillary sweating, the presence of a coarse tremor which sometimes amounted to a shivering, although they were not cold. There was a great variety of other subjective symptoms such as sleeplessness, paresthesias, perspiration, feeling of faintness or actual syncope, anorexia, diarrhea, and tremor. There was a striking disproportion between the severity and number of the subjective symptoms when compared with the objective signs of neurocirculatory asthenia. The general appearance of these invalids with their anxious faces, asthenic bearing, trembling, cold blue

(Anglican) hands, dyspnea, diffuse cardiac apical impulse, hyperesthesia of the skin or muscles of the chest wall, and the exaggeration of many of these signs and symptoms on effort, made the diagnosis easy in most instances. It should be emphasized that the diagnosis could usually be made on positive evidence, and not merely by the exclusion of organic or neuropsychiatric conditions. It was at times amazing to see these same soldiers, forced out of bed and obliged to take graduated exercises for a period of weeks, return to high grades of physical exercise and to military duty. In exceptional instances the diagnosis was doubtful for weeks, and was helped by the results of the therapeutic test of physical training under the scientific direction of Sir Thomas Lewis.

Electrocardiographic Abnormalities: Paroxysmal tachycardia, premature beats, and sinus arrhythmia are well known to occur in the course of neurocirculatory asthenia. Very exceptionally there are also prolongation of the P-R interval, changes in the QRS complex, and especially inversion of T_2 and T_3 (Graybiel and White[16]). Only recently it has been shown that pure fear can temporarily produce such changes in the electrocardiogram, resembling somewhat changes seen in coronary insufficiency.[17]

Diagnosis: (a) *Pulmonary Tuberculosis:* Here the group at Colchester undoubtedly made their greatest mistakes, for in the afterhistories of six hundred and one men followed to the fifth year by Grant,[18] twenty-two were finally diagnosed as pulmonary tuberculosis, which is an incidence 80 per cent greater than pulmonary tuberculosis in the general population of London over the same period. It must be mentioned that we were working in barracks without any X-ray machine, and it emphasizes the well-known fact that such mistakes are inevitable if routine chest X-rays are not taken.

(b) *Graves' Disease:* Contrary to what might be expected by those who still consider neurocirculatory asthenia a pre-Graves' state, the differential diagnosis is usually not very difficult. It is admitted that neurocirculatory asthenia and Graves' disease are probably related—perhaps cousins once removed—but Graves' disease has some unknown X-factor not present in neurocirculatory asthenia. The best single diagnostic criterion is of course the increased metabolic rate. In neurocirculatory asthenia the basal metabolic rate if really basal is within the normal range, as was demonstrated at Lakewood years ago by Peabody, Wearn and Tompkins.[19] Other useful diagnostic points are the general

hyperkinetic manner of the Graves' patients, the *warm* rather pink hands of Graves' disease, the persistence of tachycardia in sleep, the presence of characteristic eye symptoms, and the favorable response to iodine administration. In regions of endemic goiter, for example the northwestern part of the United States, one used to see many recruits who had the combination of simple goiter and neurocirculatory asthenia. How many of these eventually developed Graves' disease I do not know, but of the six hundred and one Colchester patients, not one developed Graves' disease within the five year period in which they were followed.[18]

(c) *Early Organic Valvular Disease:* There is real difficulty in differentiating early mitral disease and neurocirculatory asthenia. A history of rheumatic fever or chorea is not very helpful as it occurred in at least 65 per cent of the cases of mitral stenosis and in 23 per cent of the effort syndrome soldiers. A systolic murmur, accentuated first apical sound, accentuated second pulmonic sound and tachycardia may occur in either condition. One may have to examine the patients repeatedly after effort, before one is able to elicit a presystolic or diastolic murmur, a true thrill, or definite cardiac enlargement. Sometimes a cardinal physical sign, the presystolic murmur, can be brought out by auscultation immediately after exercise or by laying the patient in the supine or left lateral position. Unfortunately the symptoms of neurocirculatory asthenia are frequently superimposed upon those of organic heart disease. One should remember that neurocirculatory asthenia alone may be characterized by dyspnea, pain, palpitation, cyanosis, syncope, fatigue, dizziness—all of which also occur in heart failure, but usually not until the cardiac insufficiency is advanced.

(d) *Rheumatic Cardiac Disease:* In children one is frequently faced with the very practical problem of differentiating between a vasoneurosis with fleeting pains and perhaps fever due to some accidental complication, and true rheumatic carditis. Rheumatic fever may be diagnosed and the child sent for years at great sacrifice to the South or California. There may be all the symptoms and signs of neurocirculatory asthenia plus evidence of an infection, and still that infection may not be rheumatic fever, but a chronic pyelitis or a sinusitis. Further, even if the rheumatic state is demonstrated by definite clinical evidence such as polyarthritis, even then no permanent harm is done, unless the heart becomes involved. One should watch closely for definite evidence of

cardiac involvement, that is cardiac enlargement, a presystolic or diastolic murmur, pericarditis, or positive electrocardiographic evidence, before jumping at the diagnosis of rheumatic cardiac disease. As it is impractical to send all children suspected of rheumatic carditis for years to a climate where streptococcus infections are rare, I have tended to limit such radical advice to those who have had at least one proven attack of rheumatic infection or in whom actual rheumatic carditis has been established. There are many cases of acute rheumatic fever or chorea in which ultimately no evidence of cardiac involvement can be proven. The prophylactic treatment for rheumatic recurrences with sulfonamides is promising.

(e) *Hypertension:* In taking the blood pressure of registrants and volunteers and also of patients with neurocirculatory asthenia one encounters, not infrequently, blood pressure readings above 155 mm. of mercury for the systolic and 90 or above for the diastolic pressure. Almost every week I run across registrants with neurocirculatory asthenia and a systolic blood pressure of 180. Are these examples of essential hypertension? The majority are not, and it is probably for this reason that the examiners for the selective draft are not instructed to take blood pressures. In most instances it is the excitement which elevates the blood pressure to the figure of 150 or over. If one takes the same individual's blood pressure at home while at ease, the pressure is usually found to be within normal limits. Finding the increased blood pressure is of value in directing attention to the necessity of searching for other signs of cardiovascular or renal disease such as cardiac hypertrophy, ringing aortic second sound, or fundus changes. Paul Wood[20] using the Hines cold pressure test on twenty-five cases of neurocirculatory asthenia came to the conclusion that 90 per cent of these were not instances of incipient hypertension. Grant[18] apparently found no evidence of increased hypertension in his study of six hundred and one cases of effort syndrome followed up for five years. It is still a question in my mind whether many of these youths with labile blood pressures and a tendency to elevation, will not prove to be subjects of essential hypertension in middle life. It is important to get a more definite statistical answer to this practical question. Meanwhile it should be noted that the latest statistics of the selective draft in New York City show 2,978 rejections for persistent high blood pressure out of 115,569 registrants examined.

(f) *The differential diagnosis* of neurocirculatory asthenia from

TABLE III

AFTER-HISTORIES OF "EFFORT SYNDROME" CASES RETURNING TO CIVIL LIFE AFTER THE GREAT WAR. STATE AFTER FIVE YEARS: PERCENTAGE FIGURES (AFTER GRANT[18])

Number of cases	*Fit*	*Improved*	*Stationary*	*Worse*	*Incidence of serious disease*	*Dead*
601	15.3	17.8	56.2	3.2	8.7	2.3

anemia, angina pectoris, epilepsy (petit mal), toxic myocarditis and malingering cannot be gone into. After a year's experience in a medical advisory capacity with the selective draft, I may say that many registrants may have overemphasized their symptoms, but I have run across only one instance of deliberate malingering and that was detected.

Prognosis: The question of prognosis should be divided into (1) the immediate prognosis of soldiers invalided for neurocirculatory asthenia, based on their response to graduated physical exercise and (2) the ultimate after-war prognosis of such soldiers when they return to civil life. Under the direction of Lewis[2] the grading of men leaving the Military Hospital at Colchester was as follows:

Fit for general service	20 per cent
Fit for hardening or labor	30 per cent
Fit for light or sedentary work	30 per cent
Permanently unfit	20 per cent

This categorization generally proved to be correct as tested by the subsequent military careers of these soldiers discharged from the hospital. Our knowledge of the ultimate prognosis of soldiers after their return to civil life rests entirely on the follow-up study made by Grant[18] on six hundred and one soldiers for five years after their discharge to civil life (Table III).

Grant adds that this general prognosis is modified by the age of the patients and the tolerance to exercise. Thus the percentage of those between 17 and 20 years who were finally classified as fit was 25 per cent, whereas it was only 2.1 per cent for those between 41 and 50 years. In the same way the percentage of ultimate recoveries paralleled the grouping based on exercise tolerance, that is, the better the patient's tolerance to exercise, the greater were his chances of ultimate recovery.

You may be interested in the latest statistics of the medical findings

among registrants in the New York City area for the Selective Service Draft, for which I am indebted to Colonel S. J. Kopetzky and Dr. Arthur M. Tiber. There were 115,569 registrants examined. For diseases of the heart, blood vessels and circulation 12,261 or 10.61 per cent were rejected. For neurocirculatory asthenia 195 were rejected by the examiners, that is 1.6 per cent of the cardiovascular cases. Of course, many instances of neurocirculatory asthenia have been rejected under the caption of tachycardia (1,180 registrants) or of one of the psychoneuroses. I venture to state that many potential cases of neurocirculatory asthenia have slipped through and many of these may give adequate service without being disabled. To this day of course we do not know how many cases of neurocirculatory asthenia went through the first Great War, gave valuable service, and were never heard from by medical officers. As you are aware, neurocirculatory asthenia cases, unless mild, are entirely disqualified by the Draft Board and put into Group 4F. If mild, they are accepted in Group 1A, for full duty.

Treatment: The treatment may be divided into (1) preventive and (2) curative. The latter consists in attempts at physical and psychological rehabilitation of the individual; this is best accomplished by the collaboration of cardiologists and neuropsychiatrists.

Preventive Treatment: It is important to allow a proper convalescent period after any infection or severe operation. After complete recovery, patients should be gradually hardened before returning to full duty.

2. Neurocirculatory asthenia is due in part at least to emotional disturbance, especially fear. Therefore the cause for the emotional upset should be removed; but if this is impossible, the individual should be assisted to make an adjustment. The medical officer can sometimes stop the bullying non-commissioned officer from precipitating neurocirculatory asthenia; if not, he may adjust the recruit to this hardship and to his fear of army life.

3. Neurocirculatory asthenia should be recognized as early as possible, especially in children, and measures taken to prevent the condition from becoming chronic or permanent. In potential cases, i.e., those with family and previous personal histories of neuropsychic factors, psychic insults should be prevented or removed. The famous psychologist, William James, years ago in "A Moral Equivalent of War" advocated that every youth should serve the nation for a term of years in an industrial army. This would harden lads, teach courage and heroism, and would

probably help the younger generation to adjust themselves to life.[21]

Curative Treatment: 1. There is no specific treatment.

2. The condition should be explained at the first interview and the patient reassured that he has no organic heart disease.

3. Lewis[2] used daily graduated physical exercises with route marches over a period of five or six weeks. The results were fairly good, but it was difficult to tell whether it was the physical exercises themselves or the psychotherapeutic effect of the latter which accomplished the relatively good results. For civilians, graduated moderate physical exercise is to be recommended, such as golf, bicycling, pingpong, or swimming. The mere permission to indulge in exercise frequently convinces the patient that he has no organic cardiovascular disease.

4. Occupational therapy has made great strides since 1917, and should be of value, if only to prevent invalids from becoming depressed. Carpentry, metal work, leather work, drawing, modelling and painting will afford regular occupation for men with neurocirculatory asthenia for at least half the day.

5. Psychotherapy is indicated, but the psychotherapeutist must individualize and even then be prepared for dubious results. Attempts with psychoanalyses were not successful at the Military Hospital in Colchester, but Captain Rivers who attempted psychoanalysis admitted he could not get far with privates and believed he would have been more successful with officers. Suggestion, including hypnotism, has been used in relatively few cases which were diagnosed as hysteria. Patients should be relieved of any unusual responsibility until they have recovered.

6. Removal of foci of infection in teeth, tonsils, intestinal tract, including the appendix and gallbladder, may or may not be of service.

7. Sedatives may be used to tide over periods of insomnia or excitement. Other medication, except for individual symptoms, such as constipation, is useless. Amphetamine sulphate (benzedrine) seems to help the asthenia while it is being taken.

8. In civilian practice vitamins should be tried, although there is at present no evidence of a primary vitamin deficiency in the etiology. In almost any chronic disorder there may be a secondary clinical or subclinical vitamin deficiency, due for example to loss of appetite. Nicotinic acid has been especially vaunted for the psychoneuroses by competent neurologists. The administration of nicotinic acid in adequate doses certainly results in vasodilator phenomena which may have a beneficial

psychic effect. However, Jolliffe has given as much as 1000 mgm. of nicotinic acid a day for eight days, followed by 300 mgm. a day for a month, without making any impression on neurocirculatory asthenia. With the present army daily ration consisting of 4,500 to 5,000 calories and rich in vitamins, one would not anticipate any vitamin deficiency among soldiers,.

9. Adrenal denervation and ganglionectomy for neurocirculatory asthenia: It occurred to Crile[22] some years ago to treat those cases which are resistant to medical management by breaking the brain-adrenal-sympathetic chain at the adrenal glands by denervation, or at the celiac ganglion by ganglionectomy. "Of one hundred and fourteen cases of neurocirculatory asthenia treated by adrenal denervation and followed up, in one hundred and seven cases or 93.86 per cent, the condition was cured or improved." Of fourteen cases subjected to celiac ganglionectomy and section of some communicating branches to the adrenal medulla, thirteen were followed up; "in twelve of these, or 92 per cent the condition was improved or cured. There was only one failure." These are remarkable results.

If these therapeutic suggestions are followed, there is a fair prospect of a return to full or partial military duty of some patients, of economic restitution in civil life of others, and of symptomatic amelioration of the remainder who are permanently incapacitated for any real work. In the treatment of neurocirculatory asthenia in times of war, it is to be remembered that it is to the soldier's advantage to exaggerate and prolong his complaints. The medical officer may be torn between his obligation to the soldier as an individual and his duty to the State.

Conclusion

Neurocirculatory asthenia in its severe incapacitating form is a neurosis and usually a war neurosis frequently on a constitutional basis. It represents the somatic component of a psychoneurotic state. Its detection is now particularly important because it ear-marks those who are potential candidates for a neurosis under war conditions. It is estimated that over half of the beds in military hospitals are now occupied by patients with nervous or mental disorders,[23] thus permanently depriving others of these hospital facilities. It behooves us therefore to study this disorder, neurocirculatory asthenia more intensively, to prevent its occurrence in all the services if possible, and to learn to relieve the

condition promptly when it occurs.

It is particularly appropriate that Americans should pursue these investigations, as the classic description of the disorder was based on observations made during our own Civil War by a Philadelphia clinician, Dr. J. M. Da Costa.

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THE PRESENT STATUS OF THE DIAGNOSIS OF UNCOMPLICATED SYPHILITIC AORTITIS*

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In the last 15 years a large amount of work has been done on problems relating to cardiovascular syphilis. Some of this has been fruitful but in at least two fields considerable remains to be done, namely, in the early diagnosis of uncomplicated aortitis and in the efficacy of treatment. Our discussion will be limited to the problem of the diagnosis of uncomplicated aortitis. By this term we mean syphilitic involvement of the aorta in the stage preceding the development of aortic insufficiency, aneurysm or syphilitic coronary osteal stenosis.

In 1932 Moore[1] studied the clinical records of 105 cases of uncomplicated syphilitic aortitis which had been discovered at necropsy at The Johns Hopkins Hospital between the years 1910 and 1930. Only 4 had been diagnosed ante mortem and in only 13 others had the diagnosis been suspected. In 1927, in The Brooklyn Hospital Adult Cardiac Clinic we undertook to examine and follow all syphilitic patients enrolled in the Venereal Disease Clinic. By 1935[2] a diagnosis of uncomplicated syphilitic aortitis had been made in 59 (17.05 per cent) of the 346 patients included in the study. In contrast to this, White and Wise[3] in 1937 stated: "Since a clinical diagnosis of aortic syphilis can rarely be made within ten years after infection, it may safely be said that the early diagnosis of cardiovascular syphilis is practically impossible. If this is so, our interest is naturally next concentrated on the problem of the earliest possible diagnosis of such disease."

Because of conflicting statements, it seems desirable to attempt to throw more light upon the difficult problem of the diagnosis of uncomplicated aortitis. Since it is agreed by most pathologists that syphilis can affect the aorta soon after the primary infection, it becomes important to know in what proportion of patients this complication occurs.

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Langer[4] found that from 70 to 80 per cent of all syphilitic patients who came to necropsy at the Rudolf Virchow Krankenhaus presented evidence of involvement of the aorta. Moore, Danglade and Reisinger[1] placed the figure based on evidence collected in this country and abroad at between 80 and 90 per cent. Of course this does not imply that syphilitic aortitis was the cause of death in all these patients or that it caused cardiac symptoms. It means only that a syphilitic lesion of variable extent was present in a large per cent of all syphilitics examined post mortem. Furthermore, these statements do not take into consideration the very important factor of treatment. Vonderlehr and Usilton[5] have shown that adequate treatment, given at the time of the chancre and continued for 2 years thereafter, prevented the development of cardiovascular syphilis in a group of syphilitics observed 10 or more years after the infection. Therefore we must suspect the probability of aortitis in all syphilitics except in those who have had early and adequate treatment.

By what means can the physician arrive at a diagnosis of uncomplicated syphilitic aortitis? In the first place, he must not wait for cardiac symptoms to call his attention to the aorta because uncomplicated syphilitic aortitis is a symptomless disease. Wilson,[6] in an analysis of 211 cases of syphilitic aortitis proven at autopsy, found that cardiac symptoms were related to the development of the complications of aortitis or to some other cardiovascular lesion. If we wait until the patient comes to us complaining about his heart we will not see him until aortic insufficiency, aneurysm or coronary osteal stenosis has appeared. This statement brings to mind the symptom "aortalgia," the burning pain behind the upper sternum said to be caused by syphilitic inflammation of the aortic wall. In the fourteen years of our study we have not encountered this symptom but we have met the anginal syndrome often. It seems probable that aortalgia was the name given to the pain of coronary insufficiency before the existence of syphilitic coronary osteal stenosis was well known. If a patient with syphilitic aortitis complains of retrosternal or precordial pain on effort, we add the diagnosis syphilitic coronary osteal stenosis. However, the converse of this is not true. von Jagic and von Zimmermann-Meinzingen[7] in a clinical and necropsy study found the anginal syndrome in only 50 per cent of the patients in whom syphilis had involved the coronary arteries. Syphilitic coronary osteal stenosis may produce sudden death without previous symptoms.

In diagnosing uncomplicated syphilitic aortitis there are two physical signs that may be helpful. The first is a hollow accentuated aortic second sound. We have not been able to discover any quality in this that is characteristic of syphilitic aortitis. Hypertension and arteriosclerosis may cause the same phenomenon. Nevertheless the following rule may be helpful: In a patient under 40 without hypertension who has a hollow accentuated aortic second sound, suspect aortic dilatation due to syphilis.

The second physical sign is a systolic murmur at the aortic area. There is nothing pathognomic about this because it is also found in arteriosclerosis and hypertension but it does serve to call attention to possible changes in the caliber of the aorta or in the elasticity of its wall.

Widened retromanubrial dullness has been given as a sign of a dilated aorta. Unfortunately slight or even moderate changes in the size of this vessel cannot be detected by percussion. Only when there is great dilatation can it be demonstrated by this method. I do not mean to imply that the practice of percussion of the base of the heart and great vessels should be abandoned, because the method may detect aneurysmal dilatations, but it should not be included in the criteria for the diagnosis of uncomplicated aortitis.

Fluoroscopy, orthodiagraphy and teleoroentgenography are very useful in studying the size and shape of the aorta and should be used periodically in every patient who has had syphilis. We should never fall into the error of treating a patient with syphilis of the central nervous system, for example, for months or years and forget to have a look at his aorta in the fluoroscope. Before arriving at a judgment it is necessary to be familiar with the size, shape and position of the normal aorta. Laubry[8] has carried out some valuable studies that give much information on these points. A cadaver was suspended by the head, a radiopaque substance was injected by way of the carotid artery into the aorta and heart, thereby filling the left ventricle and auricle. Roentgenograms were taken of the heart and great vessels. The material was then washed out and a fresh supply injected by way of the superior vena cava into the right auricle, right ventricle and pulmonary artery. Roentgenograms were taken of these structures. By superimposing the two sets of films the topographical anatomy of the heart with the body in the erect position could be studied. Laubry demonstrated conclusively that in young subjects the aorta is a midline structure situated behind

the body of the sternum, rising vertically toward the neck and then curving backward and slightly to the left. The right border of the vascular pedicle is formed by the superior vena cava. As age advances, the aorta elongates, the loop widens and the ascending aorta moves to the right pushing the superior vena cava to the right at first and later backward so that in older subjects the aorta comes to form the right border of the vascular pedicle. All Laubry's work was perforce done after death when anatomical relations may be different from what they are in life. Cosseo[9] of Buenos Aires has supplied the final proof that Laubry's observations hold true for the living as well. In a patient under fluoroscopic observation he inserted a long rubber catheter through the right median basilic and subclavian veins, down the superior vena cava and into the right auricle. By this daring experiment he showed that the superior vena cava lay to the right of the aorta.

With these concepts in mind and having familiarized ourselves with the appearance of the normal aorta, the next step is to learn to recognize the characteristics of a dilated vessel. Early in our studies of cardiovascular syphilis we attempted to use the width of the vascular pedicle measured by the Vaquez method as a criterion. On the basis of Bainton's[10] work we set 7 cm. as the upper limit of normal for the vascular pedicle at any age. Whenever this measured more than 7 cm. we considered it to indicate widening of the aortic loop. Teleoroentgenograms were taken year after year on every patient. When these were reviewed in chronological order it was found that there was considerable variation in the width of the vascular pedicle in either direction in the same patient. With our present radiologic technique it is not possible to synchronize the making of the exposure with a given phase of the cardiac cycle. As a result some films are exposed when the aorta is distended by systole and others when it is contracted in diastole. Another source of error is the centering of the patient. If this is not exact there will be a change in the measurement of the vascular pedicle. The level of the diaphragm is another variable, being altered by changes in body weight or in the phase of respiration at which the film is exposed. As time went on we came to see that we could not be certain of the upper limit of normal for the width of the vascular pedicle and from this to conclude that a measurement in excess of a given figure indicated a dilated aorta. Consequently we have abandoned our original criterion of 7 cm. as the upper limit of normal and rely upon information that can be gained

through a study of the contours of the aorta in the teleoroentgenogram and under the fluoroscope. Localized bulging of the wall of the aorta, particularly to the right in the ascending portion or forward on the ventral surface can be interpreted as indicating a weakening of the wall. Under these circumstances the aorta may be said to be dilated. Again, if there is general widening of the vascular pedicle to such a degree that its right border extends as far as the right border of the heart, it is safe to assume that aortic dilatation is responsible for this, provided that abnormalities of the lungs, pleura and mediastinum have been excluded. Abnormal pulsations either localized or diffuse, if they are well marked, indicate dilatation of the aorta. When the patient is viewed in the left oblique position, the ventral surface of the normal aorta extends toward the neck in a straight line or in a gentle forward curve. A sharp bend forward indicates dilatation or elongation of the aorta.

Attempts to measure the actual diameter of the aorta, either in the ascending portion or arch, have met with failure except in those instances where advanced arteriosclerosis outlines the vessel walls distinctly. In either the right or left oblique views the luminosity of the air columns in the trachea and main bronchi make it impossible to locate the aortic walls with any degree of certainty. Kreuzfuchs[11] has described an ingenious method of measuring the true diameter of the aorta at the knob which has proved valuable in our hands. Under fluoroscopy with orthodiagraphic technique the left border of the knob of the aorta is found and marked on the screen. The patient then swallows thick barium paste which he has been holding in his mouth. As the column descends in the esophagus to the right of the aortic knob a crescentic indentation will be seen in it formed by pressure upon the esophagus exerted by the aortic arch at the knob. The central point of this crescent is then marked on the screen. The distance between these two points after 2 mm. has been subtracted for the thickness of the esophageal wall is the true diameter of the aortic knob. Roesler[12] gives 2.0 and 4.0 cm. as the minimal and maximal figures for healthy adults.

When all these methods of roentgen examination have been used, it is possible for the experienced roentgenologist to state whether there is evidence of aortic dilatation or not. However, we do not believe that it is possible for him to make a diagnosis of syphilitic aortitis on x-ray evidence alone. The clinician must decide what disease or combination of diseases is responsible for the dilatation of the aorta.

Having discussed the clinical and roentgenologic aspects of the diagnosis of uncomplicated aortitis, it is possible to derive six diagnostic criteria* therefrom:

1. The patient must be 40 years of age or younger. Ophüls[13] in a statistical survey of 3000 autopsies found that between ages 30 and 40 arteriosclerosis was slight and appeared in only 6 per cent of the cases whereas between 40 and 50 nearly one fourth of all cases showed vascular lesions. It is therefore impossible for the clinician to say whether syphilis or arteriosclerosis or a combination of both is responsible for changes in the aorta appearing after the age of forty.

2. The patient must have had syphilis beyond reasonable doubt. In our studies we have required a history of an initial infection plus one strongly positive Wassermann reaction or, if there is no history of infection, then two strongly positive Wassermann tests. If neither of these is present then there must be incontrovertible evidence of syphilis elsewhere in the body; tabes, for example. If one attempts to discover syphilitic aortitis in a group of patients in some of whom the presence of syphilis is in doubt the percentage incidence will be too low.

3. There must be no evidence of any other disease that might dilate the aorta. Arteriosclerosis, rheumatic fever, rheumatic heart disease, hypertension and hyperthyroidism must be excluded.

4. There must be roentgenographic or fluoroscopic evidence of a dilated aorta.

5. There may be a hollow, accentuated aortic second sound.

6. There may be a systolic murmur at the aortic area.

Hedley[14] has recently published almost identical criteria except that he includes "a history of cardiac insufficiency, precordial or substernal distress, or paroxysmal dyspnea" as one of them. As already emphasized cardiac symptoms appear late in the course of cardiovascular syphilis and are not helpful in the diagnosis of uncomplicated aortitis. When the results of 8 years of the cardiovascular syphilis study in The Brooklyn Hospital Cardiac Clinic were reviewed in 1935[2] the criteria for the diagnosis of uncomplicated aortitis were less rigid than at present. The age limit was 50 years and 7 cm. was used as the upper limit of normal for the vascular pedicle. Using these criteria 59 (17.05 per cent) of the 346 patients in the series were diagnosed as having uncomplicated aortitis. Now after 14 years of the study the age limit has been reduced to forty

* These criteria were first published in *The Brooklyn Hospital Journal*, April 1940.

TABLE I

DEVELOPMENT OF GRAVER LESIONS UNDER OBSERVATION

Diagnosis on First Examination	*Number of Cases*	*Developed Dilated Aorta*		*Developed Aortic Insufficiency*	*Developed Aneurysm*
		Before 40	*After 40*		
Potential	357	2*	4*	0	0
Uncomplicated Aortitis .	8			0	0
Aortitis or Arteriosclerosis of Aorta (over 40)	55			5	1

* In 2 additional cases under 40 and in 27 over 40 aortic dilatation was found by fluoroscopy but we cannot be certain that this developed during the period of observation since there had been no previous fluoroscopic examinations.

and the use of a numerical figure as the upper limit of normal for the vascular pedicle has been abandoned. As the result only 12 (2.5 per cent) of the 477 patients now included in the study are considered to have uncomplicated aortitis.

One way to test the validity of these criteria is to see whether under observation any of the patients develop the graver lesions of cardiovascular syphilis: aortic insufficiency, aneurysm or coronary osteal stenosis. None of the 12 patients (Table I) diagnosed as having uncomplicated aortitis have done so. However, there are 86 patients in the study group in whom the aorta is dilated but who are over 40 years of age. In these a diagnosis of aortitis *or* arteriosclerosis is made because it is not possible to be sure which disease is responsible for the dilatation of the aorta. Among these 86 patients, 5 have developed aortic insufficiency and one an aneurysm.

There are 357 patients diagnosed as potential cardiacs.* They have all had syphilis but when they first came to the cardiac clinic they showed no evidence of cardiovascular involvement. Two of these developed dilated aortas before they reached the age of 40 and 4 afterward. In the 2 still under 40 the diagnosis of syphilitic aortitis is justified but in those over 40 the dilatation must be ascribed either to syphilis or arteriosclerosis.

Before discussing the significance of these data, it must be stated that most of the 477 patients have been receiving antisyphilitic treatment

* According to the criteria of the New York Heart Association.

more or less regularly during their period of observation. This may be one of the reasons for the small number of patients in whom the disease progressed to the point where its late sequelae were manifest. Bruusgaard[15] in a group of syphilitic patients who had never received any antiluetic treatment found that 1.5 per cent had cardiovascular syphilis in from 3 to 10 years after infection and 10 per cent had it after 20 to 30 years.

Another explanation may be that the patients have not been observed over a long enough period. Syphilitic aortitis progresses very slowly so that its late sequelae are not discovered until an average of 20 years has passed after the initial infection. The fact that none of the patients who were under 40 when first observed developed aortic insufficiency or aneurysm while 6 of those over 40 did so seems to indicate that our failure to observe these complications in the younger group is related to the shorter duration of the disease.

Another method of testing the validity of a set of diagnostic criteria is to compare the clinical diagnosis with the findings at post-mortem examination. The difficulty with this method for our purposes lies in the fact that patients with uncomplicated aortitis do not die of this condition and therefore our opportunities for necropsy study are limited to those who have died from other causes. In the past 14 years we[16] have had this opportunity 13 times. In 4 instances in which the clinical diagnosis was "normal aorta" the necropsy diagnosis was the same in 3. In 2 cases diagnosed uncomplicated syphilitic aortitis we were right in one and wrong in the other. There were 7 patients with dilated aortas who were over 40 when this condition was discovered and because of this the clinical diagnosis was aortitis or arteriosclerosis or both. Necropsy confirmed this diagnosis in all 7 cases. It should be pointed out here that a clinical diagnosis including 2 diseases has a double chance of being confirmed post mortem. Nevertheless a group of criteria whose use makes it possible to arrive at a correct clinical diagnosis in 11 out of 13 cases appears to warrant further clinical trial.

Conclusions

1. The diagnosis of syphilitic aortitis can be made before aortic insufficiency or aneurysm has appeared.
2. The frequency of the diagnosis is not as high as that found in our first report (17.05 per cent). At present it is 2.5 per cent.

3. The measurement of the width of the vascular pedicle by the Vaquez method cannot be used as a criterion of dilatation of the aorta.
4. The diagnosis of uncomplicated aortitis should not be made in patients over 40 years of age.
5. The diagnosis "aortitis or arteriosclerosis" is a useful method of expressing the probable pathology in patients whose aortas are dilated and who are over 40 years of age.

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COAGENCY IN THE APPROACH TO THE PROBLEM OF RHEUMATIC HEART DISEASE*

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WITH but few exceptions, it is almost impossible to trace the origin of those major advances in the discovery and practical application of medical knowledge which have occurred with such rapidity in recent years. The impact of each important scientific discovery and of each demonstrated need upon a receptive mind is no longer the rare privilege of a few, but falls with similar force upon the minds of men in many places. Thus we see the simultaneous growth of otherwise unrelated movements in different parts of the world and of interest in a single problem by different groups or individuals within the same parts of the world.

For example, the fact that by last summer eleven States had, one by one, embarked upon so-called cardiac programs,[1] should not be interpreted as evidence that one State followed the pattern of another, or that they all stemmed from a common source of leadership, but rather as a normal outcropping of accumulated experiences common to persons in all these States and modified in time and form mostly by opportunity in terms of environmental differences. The fact that New York State was one of the last of these eleven, does not in itself, to my mind, single it out as being backward. In fact, quite to the contrary, one might justify the somewhat paradoxical statement that it was advanced and hence felt the need of further progress somewhat more slowly.

The New York Heart Association had developed a highly organized system of cardiac clinics and conducted valuable studies over a period of twenty years;[2] cardiac clinics of merit were in operation in two other major cities; one-half of the beds for the convalescent care of cardiac children in the country were located in this State;[3] cardiac children were extended special educational facilities, subsequently criticized

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though they have been, in New York City;[4] our medical schools and research institutions had earned important places of leadership in this field; The New York Academy of Medicine and the New York Tuberculosis and Health Association made formal recognition of the need of public education in rheumatic disease some 7 years ago;[5] and our leaders and thinkers in clinical medicine, research, and public health, had kept pace with those in other parts of the country in pointing out the needs for a comprehensive public health approach to the problem of heart disease.[2, 6-13] The foregoing, however, must be said with one sweeping reservation. Due credit must be given to the administrative imagination and genius of the minds behind the London Rheumatism Scheme,[14] which has greatly preceded, but for some unaccountable reason, very little influenced our urge to similar action.

Being thus advanced, the more the pity of course, that it took us so long to realize how inadequately we were prepared to make any appreciable inroads upon so large a problem; how insufficient the periods of hospital care being received by children with acute rheumatic fever;[2] how hopeless the chance of providing adequate convalescent care in which the whole child and his social environment were given consideration;[3] how poor our supervision of the cardiac child in school;[4] how ineffective our facilities for case finding and diagnosis; how lacking the means for coördinating and fully utilizing the services of existing agencies.

Of course these difficulties were recognized by some. The broadened concept of heart disease as a public health problem was voiced by a number of those here this evening several years before our program was undertaken. I can give no better evidence of their forethought than to quote from Dr. Boas,[15] who, in addressing the State Conference of Social Workers in 1935, said: "The State Department of Health must pay more attention to these cardiac cripples. In the past the term cripples has been restricted to children with orthopedic defects, those with paralysis following poliomyelitis or with diseases of the bones and joints. The attention of the State Department of Health has been confined to these cases. The child with heart disease is just as much a cripple as a child with a paralyzed leg and is just as deserving of rehabilitation. Emphasis of this point of view is particularly important at present when under the new Social Security Act the State Department of Health is planning a new study of the needs of crippled children. . . ."

By 1939 and early 1940, these views had grown to a point where they were well supported by medical opinion in general and Doctor Edward S. Godfrey, Jr., State Commissioner of Health, was approached by leaders from this Academy, the New York Heart Association, the American Academy of Pediatrics, two medical schools (one in New York City and one upstate), and the New York City Department of Health, with the suggestion that the time had come when the administrative assistance that the Department could best provide was much needed. This suggestion met with two coincidental and favorable circumstances; namely, the announcement by the Children's Bureau of the United States Department of Labor that certain of the Social Security funds allocated to it for Crippled Children's Services would be made available to aid in State programs for the care of children with rheumatic heart disease, and the availability of 96 beds, then unused, at the New York State Reconstruction Home at West Haverstraw, an institution under the jurisdiction of the Department of Health.

Doctor Godfrey called together two advisory groups of highly qualified physicians to consider and advise him with respect to: First, the propriety of the State's undertaking such a program; and second, the suitability of the State Reconstruction Home for a convalescent hospital service for cardiac children. Both reports were favorable to the proposed action. Accordingly, late in 1940, an administrative unit known as the Cardiac Bureau was added to the Department under the direction of my associate, Doctor David D. Rutstein, and by the early summer of 1941, the necessary personnel had been obtained and the cardiac service was opened at West Haverstraw.

The objectives of the State program and the details of the service at West Haverstraw are, I am sure, familiar to many of you since we have drawn heavily upon the membership of the Academy and the Heart Association for advice not only in the planning of which I have spoken, but also for the Advisory Committee which now guides our policies, the visiting and advisory staffs at West Haverstraw, and in other ways. Briefly, these objectives are:

1. To provide through the development of cardiac clinics (meeting the standards of the American Heart Association as adapted from those of the New York Heart Association) foci for the diagnosis of rheumatic heart disease in children and for the graduate instruction of physicians in the same;

2. To provide, through the cardiac service at the State Reconstruction Home, as ideal care as possible for children and young adults up to the age of 21 years, who reside within a reasonable distance of that institution; and otherwise, to aid communities in the organization of all of their existing agencies and facilities which might serve this objective;

3. To pursue, in coöperation with the State Medical Society, and other qualified medical groups, an active graduate medical educational program directed toward more exact diagnosis and correspondingly better handling of heart disease in children;

4. To pursue such opportunities as may present themselves for study of the epidemiology and public health aspects of rheumatic disease.

It is relevant to point out, in connection with the second objective, i.e., the provision of care, that it has never been clearly shown whether or not the specific benefits of convalescent hospital care might also be obtained by other and possibly less costly means.[13, 16] Our plan, therefore, purposes, if possible, to study the practicability and value, both from a scientific and administrative point of view, of prolonged convalescent hospital care and rehabilitation of children who present a reasonably favorable prognosis, in comparison to home care for a similar group of children who, however, will receive the benefits of qualified medical and public health nursing supervision, social supervision including improved housing and other necessary environmental adjustments, and all other benefits of which reasonable community organization is capable.

I should like, if I may, to return to the program as it relates to New York City, in which you are more particularly interested, and to which this discussion is directed primarily. Here we are confronted by a problem in which over a dozen organizations, voluntary agencies, and governmental departments, have developed a high degree of interest and, some of them, considerable activity. Obviously a common meeting ground is required, a point where the facilities, ideas, plans and problems of these agencies, all administratively independent, may be cleared and from which they may proceed to function in an efficient manner.

There are two major administrative procedures that might be applied: One, direct supervision or, as it is sometimes called, vertical administration; the other, collaboration of agencies along lines of more or less horizontal administration. Both require an administrative unit but in the former it is regulatory; in the latter, persuasive in its functions.

I wish to venture the view that for the problem at hand neither is suited. In this country, we mistrust bureaucracy. It is perhaps justifiable in an emergency where time is of major importance but ordinarily we consider it dangerous and un-American. We believe that many qualified minds form better judgments than a single mind and we are disinclined to regiment easily. On the other hand, individualism and autonomy can most certainly be carried to a fault and unless care is taken we find that such administrative autonomy tends to become the objective and attention is diverted from the fulfillment of the original purpose.

We all are acquainted with voluntary service organizations of one kind or another, some of which work splendidly with discipline and dignity, others of which fail completely. It is of the utmost importance that we find what the difference is between these and be correspondingly guided. The answer seems simple. Is it not in sincerity of purpose? If we are deeply concerned with a problem, will not small interests tend to disappear and be replaced by voluntary submission to a reasonable and sincere administrative direction? The rational approach seems to lie between the undesirable extremes of regulation, which in this case cannot be considered, and the impotence of the so-called coöperative program which so often dies with its lips still serving. In order to describe this rational approach, I have chosen the term *coagency* because, to me, it seems to connote that degree of positiveness in coöperation upon which success may well depend.

Let us, by way of illustration, inspect a few aspects of our problem and see how dependent it necessarily must be upon a functioning coagency.

First, there is the question of case finding. Here in New York City perhaps this will not present the developmental difficulties that it might in a city with less well-organized clinic facilities and provision for school medical inspection. Both are major sources. In 1934, according to Swift,[2] approximately 1800 children were discharged from New York City hospitals with varying degrees of rheumatic carditis. There is no central registry of these as in London and at present their further care is largely a matter of the affiliations of the hospital from which they were discharged, and the interest of the medical social services. Well over 5,000 cases of rheumatic heart disease are treated annually in the fifty-six affiliated clinics of the New York Heart Association.[2] Records

of these are available to the Association, but there is no mechanism for clearing them with the records of many of the hospital discharges, nor of cases detected in other ways, such as through school medical inspection, and more particularly the patients carefully diagnosed and registered by the City Department of Health through its excellent new Cardiac Classification Service.

And what if these patients, as well as those found in private practice or through other channels, were all cleared through a central point? Could the diagnosis be considered as sufficiently accurate for a registry upon which service and studies would both depend? The experiences of Paul[17] and of Rutstein[18] suggest that they cannot be so considered. For uniform standards of diagnosis, the skill of specially qualified diagnosticians appears necessary. To be sure, most creditable steps already have been made in this direction here in New York City, but means should be found for their further extension if the total problem is to be clarified.

In considering other possible methods of case finding, one cannot escape the question as to whether or not better knowledge of the epidemiology of the disease may lead, as it is doing in tuberculosis, directly to families or situations where the mass of cases may be found. The studies of Paul[19] and Gauld and his associates[20] strongly indicate this as a possibility. If it proves true, new mechanisms must be developed which will probably be a major concern of the health department.

Second, having found the patients, what is to be done? A certain proportion will need continued convalescent hospital care and gradual rehabilitation. All will require regular medical supervision for a period of years at a clinic or by their own physician. A large majority will require careful study of their home and social-environmental problems.[21] Many will require adjustments.

There are still far too few beds for the convalescent care of children with rheumatic heart disease to approach the estimated need in this city,[2] despite the recent expansion of facilities at St. Francis Sanatorium at Roslyn, Long Island, and the opening of the ninety-six bed service by the State Department of Health at its Reconstruction Home at West Haverstraw. Admission of patients from medically indigent families to these and the other available services follows different patterns varying from simple clearance through a medical research clinic into its endowed convalescent wards, to care at city expense administered by the Depart-

ment of Social Welfare, or as in the case of the New York State Reconstruction Home, care at shared City and State expense through the agency of the Domestic Relations Court and the State Department of Health.

The problem of medical supervision through clinics and private physicians is closely related to that of the follow-up observation of patients discharged from the several specialized convalescent hospitals. All of these institutions are investigative in their point of view, and are certainly entitled to any reasonable data that they may require. Yet, attempts to obtain these data must be carefully protected from falling on innumerable obstacles. It is a problem with which clinic social workers, school medical services, public health and visiting nurses, as well as physicians, will find themselves concerned.

Likewise, the problems of meeting the social-environmental and educational needs of these children cross many administrative lines and enter into the jurisdictions of several departments of government as well as being the concern of voluntary agencies.

Third, there is the question of administration. At the Federal level, the chief agency is the Children's Bureau, which, through its administration of Social Security funds for Crippled Children's Services, is in a strong position to encourage and influence the standards of State programs and of related medical care. The actual determination of policy, however, resides with the States, but New York State, under the law, has no health jurisdiction in New York City, save for its general responsibility for the expenditure of Federal Funds. I look, therefore, to the time when the City Department of Health will be in a position to incorporate certain phases of the program in the activities of its crippled children's service.

It is probably unnecessary to go further to picture the amazing amount of interlacing and the complexity of the administrative problem that faces us. Disease has often been said to have no respect for boundaries or political lines. I may add that neither does it respect man's ideas of administrative orderliness. If we are to succeed, we, as a group of agencies, must be willing to cross the preconceived lines of our several organizations where and when indicated by sound judgment. By so doing alone, can we hope to accomplish our objective with the greatest economy of facilities, manpower and expense.

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STUDIES ON HEADACHE: THE MECHANISMS AND SIGNIFICANCE OF THE HEADACHE ASSOCIATED WITH BRAIN TUMOR*

E. Charles Kunkle, Bronson S. Ray and Harold G. Wolff

Headache of patients with brain tumor has been considered to be of limited clinical interest. The mechanism of its production has been incompletely understood and its value in localization of the lesion has been minimized. This restriction of interest in the clinical importance of the headache has at least two explanations: first, the location of the pain has seemed to bear little relation to that of the tumor;[1,2,3] and second, tumor headache has been held to be a manifestation of generalized increase in intracranial pressure, and therefore of little value in localization. The observations which have been made regarding the association of headache in certain sites with specific tumor types or locations are few.[4,5,6,7,8,9,10,11] In these, the emphasis is placed upon the occipital headache characteristic of cerebellar tumors and the frontal reference of pain when such lesions produce internal hydrocephalus. The current generally accepted view of the localizing value of brain tumor headache is summarized in two sentences from a text-book of medicine written seventy-three years ago: "Pain in the head [in patients with brain tumor] is frequently limited to a circumscribed space, but the locality may not correspond to the site of the tumor. The pain, however, is generally on the side of the head corresponding to the situation of the tumor and is referred to the occiput if the tumor be situated in or upon the cerebellum."[12]

The Problem

The purposes of this study were: (1) to define the quality and intensity of brain tumor headache; (2) to ascertain in how many cases the occurrence and location of the headache could be explained and to outline the common mechanisms of brain tumor headache; and (3) to

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define when headache might be expected to have value in diagnosis and localization of brain tumor.

Patient Material

Seventy-two patients with primary brain tumor were selected. Only those patients who made adequate descriptions of symptoms were studied. In all, the location, size and type of tumor were established at operation or autopsy. The tumors were grouped as follow:

Supratentorial tumors:		
Meningioma		15
Glioma		22
Glioblastoma multiforme	13	
Oligodendroglioma	3	
Astrocytoma	2	
Angiosarcoma	2	
Ganglioneuroma	1	
Neuroblastoma	1	
3rd ventricle tumors		2
Hypophysial adenoma		8
Craniopharyngioma		7
		54
Infratentorial tumors:		
Cerebellopontile angle tumors		7
Meningioma	1	
Trigeminal "neuroma"	1	
Acoustic "neuroma"	5	
Cerebellar and fourth ventricle tumors		11
Astrocytoma	5	
Hemangioblastoma	2	
Angiofibroma	1	
Medulloblastoma	1	
Meningeal sarcoma	1	
Glioblastoma multiforme	1	
		18

Headache, either continuous or more frequently intermittent, was present in sixty-five or 9/10, and increased intracranial pressure, in forty-nine or about 7/10 of the patients. The evidence for increased pressure included one or more of the following criteria: papilledema, accentuated convolutional markings on roentgenogram of the skull, ventricular dilatation on ventriculogram, definite manifestations of pressure increase when the ventricles were tapped or the bone flap turned down, and elevated cerebrospinal fluid pressure on lumbar tap.

I. The Quality and Intensity of Brain Tumor Headache

The headache was of a deep, aching, steady, dull nature. It was not rhythmic and seldom throbbing. It was usually intermittent, but in one tenth of the patients it was continuous. The headache was sometimes

severe, but rarely was it as intense as that of migraine or the headache associated with ruptured cerebral aneurysm, meningitis, or certain febrile illnesses or that induced by certain drugs. It was usually relieved by acetylsalicylic acid, or cold packs applied to the scalp, both indications of its moderate intensity. It rarely interfered with sleep. It was aggravated by coughing, or straining at stool and sometimes it was worse in the erect than in the recumbent position. It was commonly aggravated also by the onset of a minor infection. If there were any variation in intensity during the twenty-four cycle, it was worse in the early morning.

Even when the tumor directly compressed or extensively stretched cranial nerves containing pain afferents, the pain was not equal in intensity to that of tic douloureux, and indeed was often mild or absent.[4,6,8,13]

Unless the pain were severe, nausea with tumor headache was slight. Vomiting occurred with displacement or compression of the medulla[14] and was sometimes "projectile," perhaps because it was unexpected when unaccompanied by nausea. The headache when occipital or suboccipital was sometimes associated with "stiffness" or aching of the muscles of the neck and tilting of the head toward the side of the tumor.

II. The Mechanisms of Brain Tumor Headache

Two sets of recent observations on headache mechanisms directly introduce the present study. First of all, data obtained during the operative exposure of intracranial contents have identified those structures which are pain sensitive to mechanical stimulation, and the sites of the headache thus induced.[15, 16, 17, 18] These structures are, in brief, the great venous sinuses and their tributaries from the surface of the brain, the dural arteries, the internal carotid arteries, the cerebral arteries at the base, the basilar and vertebral arteries, the other arteries near their sites of origin from the basilar and vertebral trunks, parts of the dura at the base, and the intracranial portions of the trigeminal, glossopharyngeal, vagus and upper cervical nerves. It was noted, furthermore, that stimulation of the pain sensitive structures on or above the superior surface of the tentorium cerebelli resulted in pain transmitted by the fifth nerve and located in regions on the anterior half of the head.

Stimulation of the pain sensitive intracranial structures on or below the inferior surface of the tentorium cerebelli resulted in pain over the posterior half of the head, the pain pathways being chiefly in the ninth

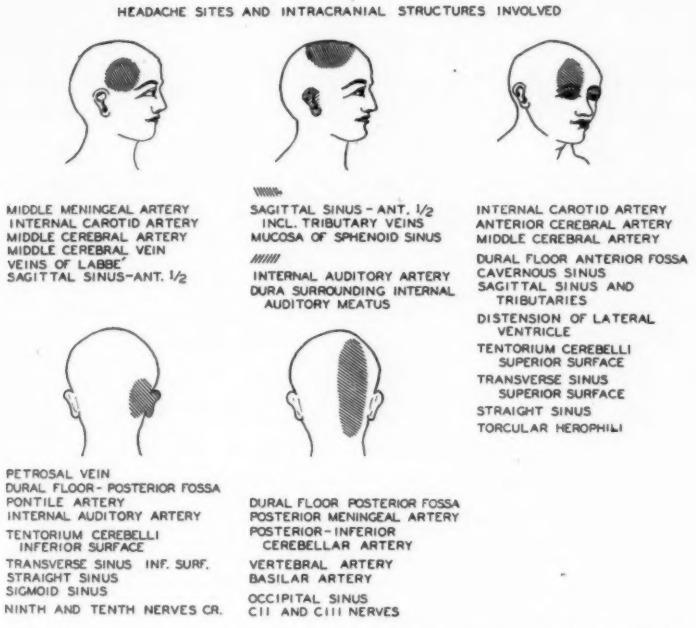

Fig. 1—Summary of intracranial pain sensitive structures which may be the sources of headache in the various main divisions of the head. Chart is based upon data obtained at operative exposure of intracranial contents.

and tenth cranial nerves and the upper three cervical nerves.[18]

These facts of pain reference are illustrated on a chart (Fig. 1) representing the sites of intracranial disease which may account for headache in the various main divisions of the surface of the head.* The chart demonstrates the difficulties inherent in any attempt to use headache as a localizing sign of intracranial disease, since lesions of remotely separated structures within the skull may cause headache in identical areas.

Organization of the data available from these studies has indicated six basic mechanisms of headache, involving distortion or inflammation of intracranial pain sensitive structures.[18]

1. Traction on the large venous sinuses or their tributaries from the surface of the brain.
2. Traction on the middle meningeal artery.

* Eye and paranasal sinus diseases have been eliminated from consideration here. The role of these factors in headache is now being studied.

3. Traction on the large arteries at the base of the brain.
4. Direct pressure upon the cranial and upper cervical nerves which carry pain fibers.
5. Dilatation of intracranial arteries.
6. Inflammation in or about any of the intracranial pain sensitive structures.

It has been suggested that the first four of these six mechanisms may play a part in the headache associated with brain tumor.[18]

A second fact important to an understanding of brain tumor headache is that increased intracranial pressure is not essential to its production.[19] Thus, elevation of the intracranial pressure in normal human subjects to levels as high as 510 mm. of saline by the intrathecal injection of normal saline consistently failed to cause headache. Evidence even more compelling that increased intracranial pressure and tumor headache are not closely related was obtained in the study of a man with a left parietal oligodendroglioma in whom bifrontal headache had been present intermittently for two months but happened to be absent at the time the following experiment was performed.[19] By drainage of cerebrospinal fluid with the patient horizontal, moderate headache, chiefly left frontal, was induced. The headache was then relieved at once by restoration of fluid and elevation of pressure to its initial level, and furthermore could not be produced by pressure elevation to a high level, 550 mm. of saline. (See also Northfield[10] and Pickering.[20])

1. *Tumor Headache and Increased Intracranial Pressure not Essentially Related:*

In this series of seventy-two patients, the symptom headache occurred almost as commonly in those patients (19 of 23, or about 8/10) without increased intracranial pressure as it did in those (46 of 49, or about 9/10) with increased pressure. Moreover, of the seven patients (about 1/10 of all cases) who were headache-free, three had increased intracranial pressure.

These data demonstrate that increased pressure per se is neither an essential nor a major factor in tumor headache.

2. *Tumor Headache in Patients with Normal Intracranial Pressure—The Mechanism of Local Traction:*

Of the twenty-three patients with normal intracranial pressure, nineteen had headache as a symptom. In all but three patients with hypophysial adenoma, the existence and location of the headache could be

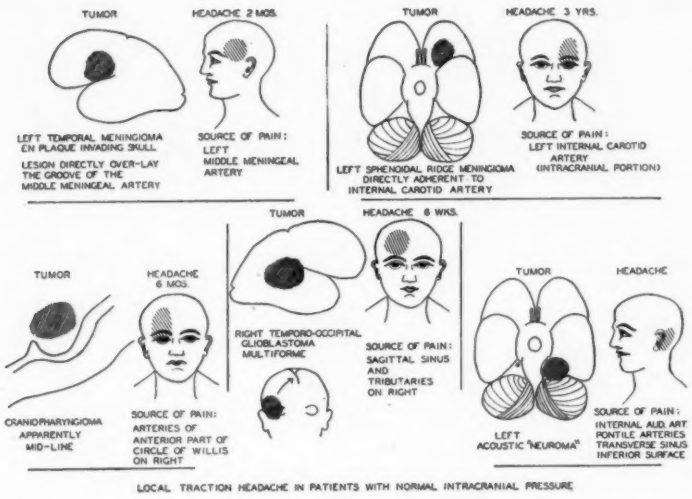
TUMOR
HEADACHE 2 MOS.
LEFT TEMPORAL MENINGIOMA EN PLAQUE INVADING SKULL
LESION DIRECTLY OVER-LAY THE GROOVE OF THE MIDDLE MENINGEAL ARTERY
SOURCE OF PAIN: LEFT MIDDLE MENINGEAL ARTERY
TUMOR
HEADACHE 3 YRS.
LEFT SPHENOIDAL RIDGE MENINGIOMA DIRECTLY ADHERENT TO INTERNAL CAROTID ARTERY
SOURCE OF PAIN: LEFT INTERNAL CAROTID ARTERY (INTRACRANIAL PORTION)
TUMOR
HEADACHE 6 MOS.
CRANIOPHARYNGIOMA APPARENTLY MID-LINE
SOURCE OF PAIN: ARTERIES OF ANTERIOR PART OF CIRCLE OF WILLIS ON RIGHT
TUMOR
HEADACHE 6 WKS.
RIGHT TEMPORO-OCCIPITAL GLIOBLASTOMA MULTIFORME
SOURCE OF PAIN: SAGITTAL SINUS AND TRIBUTARIES ON RIGHT
TUMOR
HEADACHE
LEFT ACOUSTIC NEUROMA
SOURCE OF PAIN: INTERNAL AUD. ART PONTILE ARTERIES TRANSVERSE SINUS INFERIOR SURFACE
LOCAL TRACTION HEADACHE IN PATIENTS WITH NORMAL INTRACRANIAL PRESSURE

Fig. 2—Five examples of patients with tumor headache produced by local traction. Intracranial pressure was normal in all. The structures which were the probable sources of the pain are listed in each case.

explained by traction upon or distortion of directly neighboring pain sensitive structures, and in some at operation the headache was thus reproduced. These adjacent structures were: (1) for the four supratentorial meningiomas—the superior sagittal sinus and its tributaries, the middle meningeal artery and the large arteries at the base; (2) for the one glioma—the superior sagittal sinus and its tributaries; (3) for the three craniopharyngiomas—the large arteries at the base; (4) for the four hypophysial adenomas—the large arteries at the base and in one patient the lining of the sphenoid sinus; and (5) for the four cerebellopontile angle tumors—the internal auditory artery, the pontile arteries, the dura about the internal auditory meatus, and the transverse sinus. In Fig. 2 are shown examples of this group.

Another example of the headache produced by local traction may be cited (Fig. 3). A thirty-seven year old male with a trigeminal "neuroma" in the right cerebellopontile angle and no evidence of increased intracranial pressure had had for four months three separate kinds of head discomfort. These, with the sites of origin of the symptoms, were: (1) right temporal "tightness"—fifth cranial nerve; (2) "soreness" in the

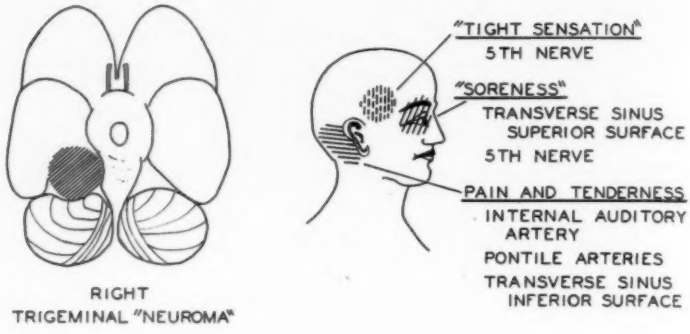

Fig. 3—Demonstration of local traction headache mechanisms in an adult male with three separate types of head discomfort, due to a trigeminal "neuroma" in the right cerebellopontile angle. The intracranial pressure was normal.

right eyeball, superior surface of the right transverse sinus and fifth cranial nerve; (3) pain and tenderness in the right postauricular area, right internal auditory and pontile arteries and inferior surface of the transverse sinus.

This mechanism of tumor headache may be termed local traction. While independent of increase in intracranial pressure, it may well be augmented by generalized displacement of the brain associated with cerebrospinal fluid pressure rise.

The three patients with hypophysial adenoma in whom local traction did not entirely account for the headache had pain in the occiput or suboccipuit in addition to frontotemporal headache. The pain in the back of the head in these three of the seven patients with hypophysial adenoma is unexplained.

3. *Tumor Headache in Patients with Increased Intracranial Pressure —The Mechanisms of Local and Distant Traction:*

In less than one-half (18) of those patients (46) with headache and increased intracranial pressure could the locations of the headache be explained or the pain induced by local traction. Most of the remaining half of the patients had posterior head pain in association with supratentorial tumors and frontal headache in association with infratentorial

tumors. The following analysis of the group illuminates this seeming paradox.

A. DISTANT TRACTION THROUGH EXTENSIVE DISPLACEMENT OF THE BRAIN:

Slightly less than one-half (14 of 32) of the patients with supratentorial tumor with headache had pain in the posterior half of the head—occipital, suboccipital or postauricular areas. In seven patients this pain was bilateral. In the seven in whom it was unilateral it was homolateral to the tumor in all but one. In each instance pain was also present in one or several other head regions.

Headache so located in these patients could not be explained by local traction, for there is no evidence that any supratentorial structure can be the direct source of posterior head pain. Pressure downward upon the tentorium cerebelli from above has been shown experimentally to cause only fronto-orbital pain,[18] probably by traction upon the upper surfaces of the transverse sinuses. Such a mechanism, therefore, cannot be relevant to posterior head pain. It has been recognized, however, that when supratentorial lesions are large enough to cause generalized increase in pressure, there is often a widespread shift in the brain causing distortion of supra- and infratentorial structures. Thus, traction or pressure upon the transverse and occipital sinuses, the basilar and vertebral arteries, the ninth and tenth cranial and upper cervical nerves is probably a common occurrence. However, with supratentorial tumor the extent of such distortion in the posterior fossa is much less and the establishment of a "cerebellar pressure cone" is less likely than in patients with infratentorial tumor.

Herniation of the hippocampal gyri through the incisura tentorii may also be responsible for posterior head pain from supratentorial tumors. In a recent review of autopsied cases of supratentorial neoplasms such a complication was found in about 8/10 of the series.[21] The distortion produced by this "temporal pressure cone" has been shown to affect not only the hippocampal gyri but also the adjacent brain stem, and presumably the basilar artery and its branches. This mechanism must be considered to be of minor importance as regards the present problem, for in the autopsied series reported, posterior head pain was no more common in the patients with such herniation than in those without it.

In brief, posterior head pain in patients with supratentorial tumor appears to depend upon expansion of the mass to such an extent that

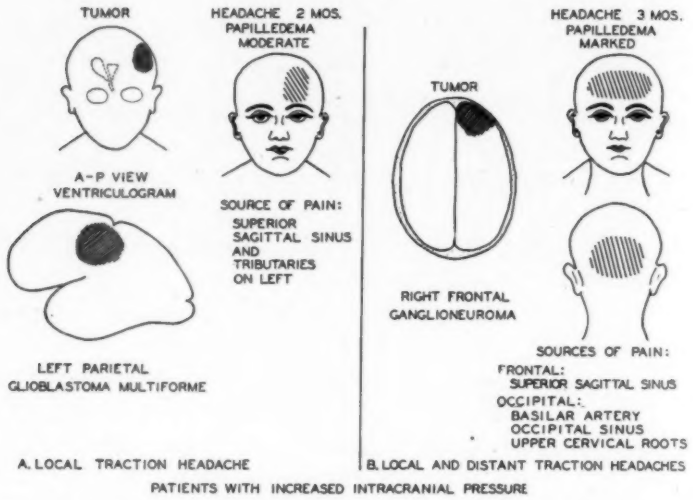

Fig. 4—Local and distant traction mechanisms in tumor headache contrasted in two patients with increased intracranial pressure. In A the headache was due to local traction alone; in B the frontal headache was due to local traction, the occipital headache to distant traction.

coincident with generalized rise in intracranial pressure, traction is produced upon pain sensitive structures in the distant posterior fossa, by displacement of the whole brain. In contrast to local traction this may be conveniently termed distant traction and may be considered as a second mechanism of brain tumor headache. It is evident that in extensive shift of the brain, traction effected at a distance from the tumor may involve structures on the same side as the tumor or both sides. The data indicate that both situations are common.

Two cases of supratentorial tumor with increased pressure are illustrated in Fig. 4A and B. In A, a patient with a left parietal glioblastoma multiforme and moderate papilledema, the left frontal headache was due to traction locally by the mass upon the superior sagittal sinus and its tributaries on the left. The ventriculogram is reproduced in outline to emphasize the type of distortion which was present. In B, a patient with a right frontal ganglioneuroma and advanced papilledema, the frontal headache was similar in its mechanism to that of A. The occipital headache represented distant traction upon structures in the posterior fossa, secondary to widespread displacement of the brain.

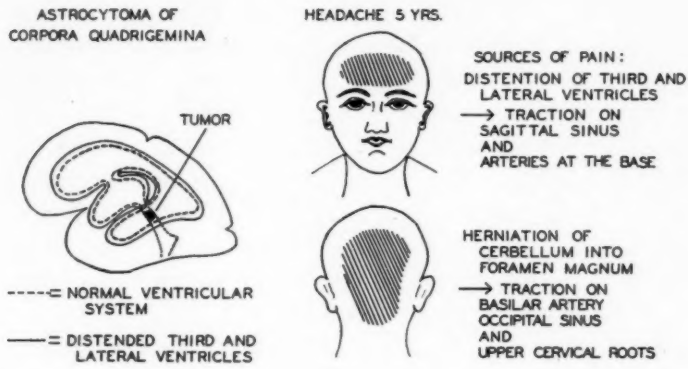
ASTROCYTOMA OF
CORPORA QUADRIGEMINA
TUMOR
-----= NORMAL VENTRICULAR SYSTEM
——= DISTENDED THIRD AND LATERAL VENTRICLES
HEADACHE 5 YRS.
SOURCES OF PAIN:
DISTENTION OF THIRD AND LATERAL VENTRICLES
→ TRACTION ON SAGITTAL SINUS AND ARTERIES AT THE BASE
HERNIATION OF CERBELLUM INTO FORAMEN MAGNUM
→ TRACTION ON BASILAR ARTERY OCCIPITAL SINUS AND UPPER CERVICAL ROOTS
DISTANT TRACTION VIA INTERNAL HYDROCEPHALUS AND CEREBELLAR "PRESSURE CONE"

Fig. 5—Demonstration of distant traction headache produced by a small astrocytoma in the roof of the aqueduct. The frontal headache was due to internal hydrocephalus, the occipital headache to distortion of posterior fossa structures by wedging of the cerebellum into the foramen magnum.

B. DISTANT TRACTION THROUGH INTERNAL HYDROCEPHALUS:

Frontal headache was a symptom in two-thirds (10 of 14) of the patients with infratentorial tumor and increased intracranial pressure, all of whom had headache. This fact likewise required explanation in terms other than local traction by the tumor. Analysis revealed that 9 of the 10 with frontal headache, comprising 2 angle tumors and 7 cerebellar or fourth ventricle tumors, were complicated by block to the cerebrospinal fluid outflow at the aqueduct or the fourth ventricle and had internal hydrocephalus with increase in intracranial pressure. The frontal headache was bilateral in all but one patient.

The association of internal hydrocephalus with frontal headache in patients with infratentorial tumor has been noted by others.[5, 8] Direct evidence that the hydrocephalus is causally related to the headache has been provided by the demonstration that distention of one lateral ventricle with a balloon at operation induces homolateral frontal headache[18] by traction upon the veins over the convexity of the brain anchoring it to the superior sagittal sinus. Experimental distention of the third ventricle has been found to cause diffuse headache arising from traction upon the many large vessels at the base. There is further evidence that

significant distortion of adjacent structure at the base is produced by enlargement of the third ventricle in internal hydrocephalus, for in such patients a visual field defect indicative of chiasmal pressure has often been found.[22] The usual bilaterality of the frontal headache in these patients may therefore be understood.

Hence, frontal headache of this kind is like the posterior head pain discussed in the previous section in that it is produced by traction at a distance from the tumor.

An example of a small tumor producing headache entirely by distant traction is outlined in Fig. 5. A sixteen year old male had intermittent bifrontal and bioccipital headache for five years. On admission moderate papilledema was noted. The ventriculograms showed advanced dilatation of the third and lateral ventricles. Exploration confirmed the presence of internal hydrocephalus. The foramina of Monroe were widely stretched, the largest diameter of the left opening measuring 2.5 cm. Moreover, as seen in a second exploration, the tonsils of the cerebellum were herniated in part into the foramen magnum. No tumor could be found, but autopsy revealed a small fibrillary astrocytoma of the corpora quadrigemina occluding the aqueduct of Sylvius. The frontal headache was due to distention of the lateral, and possibly the third, ventricles. The occipital headache was due to traction upon the basilar artery, the occipital sinus, the ninth and tenth cranial nerves and upper cervical roots by the wedging of the cerebellum down into the foramen magnum. The site of origin and the location of both frontal and occipital headaches were remote from the tumor itself.

C. TUMOR HEADACHE UNEXPLAINED BY LOCAL OR DISTANT TRACTION:

In two patients with increased intracranial pressure the headache could not clearly be related to these mechanisms. One, a twenty-four year old female, had a left temporal headache for five months. She had a right acoustic "neuroma," increased intracranial pressure and internal hydrocephalus.

The other, a forty-four year old female, had right occipital headache for six months. She had a left cerebellar hemangioblastoma, increased intracranial pressure and internal hydrocephalus.

Headache in this pattern, sparing the homolateral side but involving the contralateral side, appears bizarre. The direction and distribution of the stress and strain in these two cases is conjectural and the factors

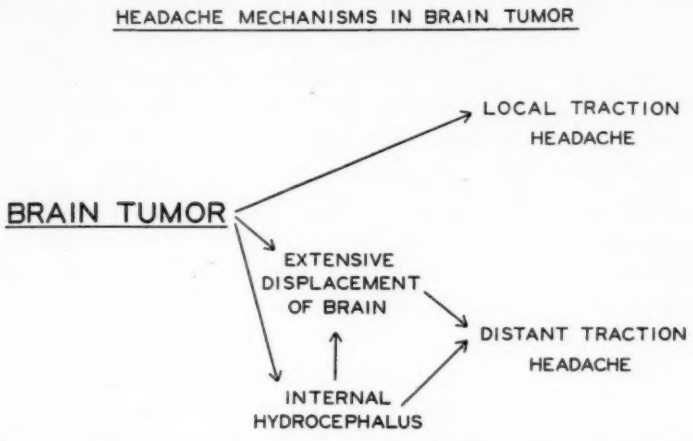

Fig. 6—Schematic outline summarizing common mechanisms of brain tumor headache.

which make them atypical are not apparent. There is no evidence, however, that mechanisms other than traction upon pain sensitive structures are involved.

Thus, in forty-four of the forty-six patients with headache and increased intracranial pressure the existence and location of the headache could be accounted for on the basis of the mechanisms thus far outlined. In many (18) local traction alone appeared to be responsible. In a few (4) traction at a distance alone was responsible, either through displacement of posterior fossa structures in patients with supratentorial tumors or through internal hydrocephalus in patients with infratentorial tumors, but, in the largest group (22) both local and distant traction were concerned. In 2 patients the headache could not be clearly explained by either mechanism.

It is to be emphasized that the association of increased intracranial pressure with headache due to distant traction does not justify the inference that increased intracranial pressure of itself causes headache. It indicates rather that the same factors which bring about distant traction, that is, extensive displacement of the brain directly by the tumor or indirectly by internal hydrocephalus, will also cause generalized elevation of intracranial pressure. (See Fig. 6.)

4. Continuous vs. Intermittent Headache with Brain Tumor:

As mentioned above, the headache of brain tumor is usually inter-

mittent. In this series continuous headache was noted in only seven cases or about a tenth of the patients (all with supratentorial tumor). Four of these had increased intracranial pressure. Local traction appeared to be the mechanism in all seven cases. The persistence of the headache in these cases is not surprising, for it is reasonable to expect local traction to be continuous, and in fact progressive, if the tumor growth is moderately rapid. There is no explanation of the intermittency of the headache that will apply to all the remaining large group of patients. In some, intermittent block to cerebrospinal fluid outflow either because of varying states of brain hydration or by movable tumors in the 4th or 3rd ventricles appears to have been responsible. In others the increase in intracranial venous pressure with straining and coughing might have been the basis for occasional headache.

5. *Patients with Brain Tumor without Headache:*

Seven patients, or about a tenth of the entire series, had no headache in association with their lesions. The tumors represented were all supratentorial; in three of the seven increased intracranial pressure was present.

Associated with increased intracranial pressure and without headaches were:

Tumor Type	*Site*
Meningioma	left parasagittal parietal
Glioblastoma multiforme	right frontocallosal
Glioblastoma multiforme	left frontal (subcortical)

Associated with normal intracranial pressure and without headaches were:

Tumor Type	*Site*
Meningioma	right frontotemporal (invading the skull)
Oligodendroglioma	left frontotemporal
Oligodendroglioma	right temporoparietal
Craniopharyngioma	suprasellar (sella and dorsum destroyed)

Review of these patients disclosed that the only characteristic common to all was a supratentorial location of the tumor.

The absence of headache in patients with brain tumor may be related to the slow growth of the neoplasm. Thus five of the above seven patients had lesions which are notably slow in expanding, i.e., meningioma, craniopharyngioma and oligodendroglioma. Moreover, in one of the two patients who were headache-free with glioblastoma multiforme, ordinarily a rapidly growing neoplasm, the clinical course indicated unusually slow growth of the tumor. When expansion of the tumor mass is slow, mechanical adaptation of adjacent structures may be sufficient to

prevent pain production. This may represent the return of distorted pain endings to normal contour even though gross distortion of the tissues in which the pain endings are embedded persists. When the pace of growth is fast, such adaptation may be inadequate.[6, 13]

It may be noted that in contrast to tumors above the tentorium, posterior fossa tumors, whatever their rate of growth, rarely fail to cause headache, undoubtedly because many of these lesions cause internal hydrocephalus.[5, 8] In consideration of headache the speed of growth of the posterior fossa tumor is less significant since it bears no direct relation to the completeness of occlusion of the iter or the fourth ventricle. A block once induced results in a relatively sudden displacement of the brain and stimulation of local and distant pain sensitive structures.

Serious sensorial defects produced by the tumor, particularly with frontal lobe lesions, may be a second cause for absence of headache. For example, in the second of the two patients with glioblastoma multiforme without headache, listed in the table above, the tumor was in the right frontocallosal region and apathy and confusion were early and dramatic symptoms. Under such circumstances, gross defects in reaction to pain may veil or completely mask headache.

Invasion of the skull by meningiomas may be still a third factor in preventing or delaying headache. The bone of the skull is insensitive and extension in this direction may take the place of greater intracranial expansion toward pain sensitive structures.

6. Consideration of Other Suggested Mechanisms.

In these formulations distortion of pain sensitive structures has been described as the chief basis for brain tumor headache. Factors other than traction have been suggested by others but not established as relevant mechanisms. Dilatation of intracranial veins and arteries, which occurs secondary to generalized increase in intracranial pressure, may conceivably be a cause of headache.[10, 18] Such vasodilatation must, however, be of minor importance as a cause of headache in patients with tumor since it has been shown in this discussion that increased intracranial pressure is not essential to the headache, and the pain in these patients is usually localized rather than diffusely distributed as it should be if generalized dilatation of intracranial vessels were the cause. Stretch of the dura mater over the convexities of the cerebrum and cerebellum by the rise in intracranial pressure is also probably not a significant mechanism, because the dura and pia in these areas are insensitive.

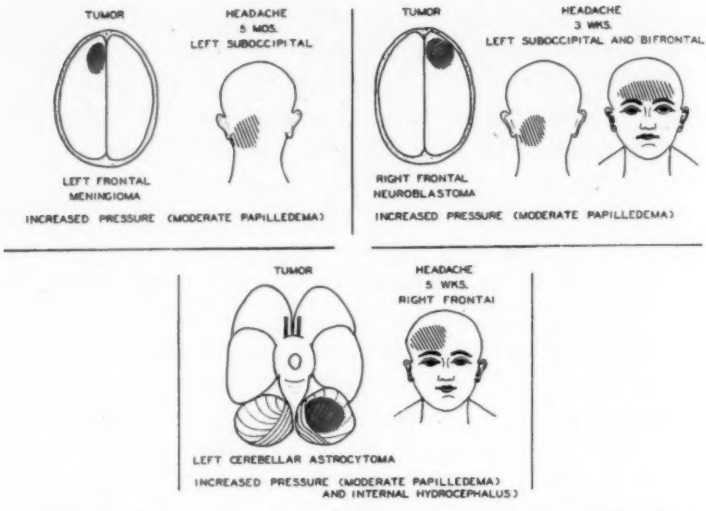

Fig. 7—Demonstration of headache as a grossly misleading sign of the location of brain tumor when it is due to distant traction. Extensive displacement of the brain and increased intracranial pressure were present in all three patients.

III. Headache in the Localization of Brain Tumor

A. Limitations of Headache as a Localizing Sign:

1. Headache in Association with Increased Intracranial Pressure of Limited Localizing Value:

In the preceding section it was noted that headache in patients with normal intracranial pressure could, with rare exceptions, be explained entirely in terms of local traction. Of patients with increased intracranial pressure in only eighteen of forty-six, or about 4/10, was local traction solely responsible; in the remaining twenty-six distant traction was an accessory or the chief mechanism.

When distant traction is involved the problem of localization of the tumor is complicated, for with this mechanism distortion of bilateral pain sensitive structures is common, and the site of the expanding mass is often concealed. It is evident, therefore, that in the presence of increased intracranial pressure, which in patients with brain tumor is

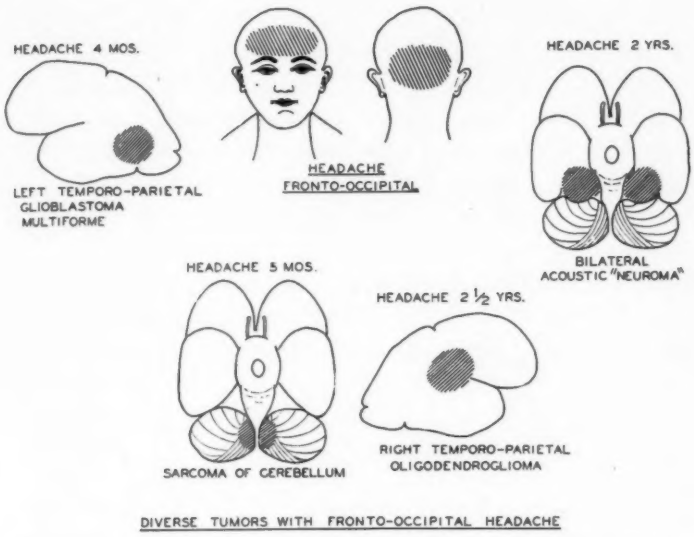

Fig. 8—Demonstration of fronto-occipital headache occurring with tumors of widely varying types and locations and therefore of no localizing value. Extensive displacement of the brain and increased intracranial pressure were present in all.

presumptive evidence of extensive displacement of the brain, the localizing value of tumor headache becomes reduced. Examples of patients with headache as a misleading sign when secondary to distant traction are shown in Fig. 7.

2. *Fronto-occipital Headache of No Localizing Value:*

The combination of frontal and occipital headache was noted in one-quarter of all the patients with headache. The group with fronto-occipital headache included patients with meningioma, cerebral glioma, third ventricle cyst, hypophysial adenoma, cerebellopontile angle and cerebellar and fourth ventricle tumors. Except for the two instances of hypophysial adenomas, all were accompanied by increased intracranial pressure. Internal hydrocephalus was present with only one of the supratentorial tumors, the 3rd ventricle cyst, but was present with all five infratentorial tumors.

The data indicated that fronto-occipital headache occurred almost as frequently with supratentorial tumors as with those below the ten-

torium. Moreover, they showed conclusively that the fronto-occipital headache combination cannot be considered diagnostic of internal hydrocephalus in patients with brain tumor. It is of additional interest that except for the hypophysial adenomas all of the patients with headache of this pattern had increased intracranial pressure.

Four typical instances of brain tumor of contrasting types and location, all presenting fronto-occipital headache, are outlined in Fig. 8.

3. Headache Over the Vertex—Infrequent and of Little Localizing Value:

Headache over the vertex was noted infrequently in this series. In only three patients was the headache in the vertex alone; these were a mid-line olfactory groove meningioma, a craniopharyngioma and an eosinophilic hypophysial adenoma. In the first two the intracranial pressure was elevated. In two other patients headache was vertical in part but not predominantly; the tumors found were a parasagittal parietal meningioma and a mid-line cerebellar astrocytoma, both with increased pressure.

4. Headache from Mid-line Tumors Not Always Bilateral:

Twenty-three mid-line tumors with headache were included in this series. In seventeen patients the headache was symmetrically bilateral, eleven having increased intracranial pressure. In six the headache was entirely or predominantly unilateral; none of these had increased intracranial pressure.

Local traction apparently was the mechanism for the headache when it was unilateral. The inequality of the distortion produced may have been due to unrecognized asymmetrical expansion of the growing tumor. Whatever the explanation, it is clear that unilateral headache may occur with mid-line tumors.

B. THE ASSETS OF HEADACHE AS A LOCALIZING SIGN:

The limitations of headache having thus been considered, its value may now be appraised.

1. Headache Near or Overlying the Tumor:

Headache to be of service as a localizing sign of brain tumor must be interpreted in terms of the headache mechanisms and principles of pain reference just outlined. Only when tumor headache is caused solely by local traction is it of direct value in localization. Even then the headache may not overly the tumor, for in about 4/10 of the patients with

headache due only to local traction the pain was not immediately over the tumor. In the following analysis certain tumors located at the base and anteriorly were not included since obviously they could not produce headache near to or directly overlying the tumor, other than in the nasopharynx. These lesions were the craniopharyngiomas, hypophysial adenomas, a suprasellar meningioma, and a third ventricle cyst. The evidence in summary, based on the remaining 22 patients with local traction headache, was as follows:

	Headache was near to or overlying the Tumor
Supratentorial Tumors	
Meningioma	3 of 6
Glioma	4 of 9
Infratentorial Tumors	
Cerebellopontile angle tumors	4 of 4
Cerebellar and 4th ventricle tumors	2 of 3
	13 of 22

When the analysis was broadened to include all the patients (again excluding the tumors at the base) headache was found to overly the tumor in only nineteen of fifty-one, or about one-third.

2. Headache as First Symptom of Brain Tumor:

With the exception of the cerebellopontile angle tumors, the presence of most of the posterior fossa tumors first was made manifest by headache. The tumors above the tentorium, on the other hand, were more likely first to cause other symptoms, as visual disturbances, paresthesias, convulsions, sensorial and personality changes. Other authors have reported similar observations.[3, 6, 10] Northfield's figures[10] based upon a series of 100 cases are presented in brackets for comparison with the data of this New York Hospital series. Headache as first symptom of brain tumor:

		Northfield[10]
In 54 cases of supratentorial tumor		
Meningioma	5 of 15	[36 per cent]
Glioma	11 of 22	[36 per cent]
3rd ventricle tumors	1 of 2	
Hypophysial adenoma	1 of 8	
Craniopharyngioma	2 of 7	
	20 of 54 or about one-third	
In 18 cases of infratentorial tumor		
Cerebellopontile angle tumors	1 of 7	[0]
Cerebellar and 4th ventricle tumors	9 of 11	[83 per cent]
	10 of 18	

The usual occurrence of headache as first symptom in patients with cerebellar or fourth ventricle tumors is related to the fact that internal

hydrocephalus can easily result from such lesions. Angle tumors, however, are less frequently associated with internal hydrocephalus in the early stages and are directly adjacent to the several cranial nerves which traverse the angle region, potential sources of such symptoms as facial paresthesias, deafness, tinnitus, or weakness of face or jaw. Whether or not supratentorial tumors cause focal symptoms or signs before headache appears depends upon the region of the brain involved.

3. *The Significance of Headache in the Back of the Head and Neck with Brain Tumor:*

Pain in the back of the head—occipital, or postauricular or suboccipital areas—was present alone or in part in one-half of the patients.* As described in a preceding section, such pain was present in seventeen of the forty-seven patients with supratentorial tumor with headache. In each of these, headache was present also in one or several other areas on the anterior half of the head. Except for the patients with hypophysial adenoma, headache in all was accompanied by increased intracranial pressure.

In contrast, pain in the back of the head was present in almost all of the patients with infratentorial tumor. Of the eighteen patients sixteen or almost 9/10 had posterior headache. The two without such headache had respectively an acoustic "neuroma" and a cerebellar astrocytoma, both accompanied by increased intracranial pressure.

It seems generally true, therefore, that occipital, suboccipital or postauricular pain did not of itself identify a tumor as being above or below the tentorium. On the other hand, the evidence did indicate that when posterior headache was absent the tumor was rarely infratentorial.

4. *The Significance of Headache in the Front of the Head with Brain Tumor:*

Frontal headache with brain tumor in this series was even more common than was pain in the back of the head. In 6/10 of all patients with headache, frontal headache was present, alone or with headaches elsewhere. In contrast to pain in or near the occiput, frontal headache was noted somewhat more often in association with supratentorial tumors (32 of the 47 with headache or about 7/10) than with infratentorial tumors (10 of 18 or about one-half).

Pain solely frontal was noted in eleven of the forty-seven cases of supratentorial tumor with headache but in only two of the eighteen

* For the sake of brevity pain of this type will be referred to hereafter as posterior headache.

cases with infratentorial tumor or headache.

The predominance of frontal headache is not surprising in view of the diverse ways, direct and indirect, in which brain tumor may produce pain referred to the frontal areas. But from the data it may be inferred that although frontal headache was common in all types and locations of tumor, the pain when solely frontal was usually due to supratentorial tumor.

5. *The Significance of Unilateral Headache:*

It has been shown above that mid-line tumors may cause unilateral headache, probably because of asymmetrical expansion of the mass. Further data concerning the significance of unilateral headache were derived from an analysis of the forty-two remaining patients with headache and tumor not in the mid-line. The headache patterns were as follows:

Headache homolateral to the tumor	24
Headache contralateral to the tumor	5
Headache symmetrically bilateral	13

All five patients with headache contralateral to the tumor had increased intracranial pressure. In three of these five the contralaterality of the headache was the result of distant traction.

It is evident that headache chiefly on one side of the head was common in these patients, and that in most cases the headache was on the same side as the tumor (See also Pickering[20]). Contralateral headache may have resulted from the presence of anatomical asymmetries in the brain or skull such as the size or position of the lateral or third ventricles, or the relative position of the cerebellum, brain stem and anchoring structures in the posterior fossa. In none of the patients with normal intracranial pressure was contralateral headache noted. The important inference from these considerations is that headache solely or chiefly unilateral is probably on the same side as the tumor when the intracranial pressure is normal.

C. VARIOUS TYPES OF TUMORS IN RELATION TO HEADACHE:

Among patients with craniopharyngiomas, headache as an initial symptom was rare and its location unpredictable. The hypophysial adenomas also rarely produced headache as an initial symptom, and the site of the headache presented nothing of specific or helpful value.

Among most of the patients with headache due to cerebellopontile angle tumors the headache was a leading clue in the localization of the

lesion, although headache did not occur as an early symptom. Headache solely in the postauricular area was almost specific, especially before gross displacement of the brain occurred.

Among patients with meningiomas (all but one being supratentorial), headache was a first symptom in one-third. In about one-half of the fourteen patients with headache the headache was predominantly unilateral and on the same side as the tumor, and in all but two of the fourteen cases the headache was due to local traction. It might be expected that situated as the meningiomas are, in contact with pain sensitive structures at the base and over the convexities, that headache as an initial symptom might occur in a higher proportion than the one-third indicated. It is probable, however, that the slow growth and bone invasion offset the effects of proximity to pain sensitive vascular structures.

Gliomas, despite their lack of direct contact with pain sensitive structures, presented headache as a first symptom in one-half of the patients when they occurred above the tentorium and even more frequently when they occurred below. Such tumors produced headaches as an early manifestation probably because of their speed of growth and the likelihood of their occluding the lateral, third, and fourth ventricles.

D. GENERALIZATION:

From these studies the following generalizations concerning brain tumor headache as an aid to localization seem justified.

1. Although the headache of brain tumor is often referred from a distant intracranial source, it approximately overlies the tumor in about one-third of all patients.

2. Brain tumor headache in the absence of papilledema is of great localizing value. In about two-thirds of such patients the headache immediately overlies or is near the tumor and in all when unilateral it is on the same side as the tumor.

3. Headache is almost always present with posterior fossa tumor.

4. Headache may be absent with any of the common types of supratentorial tumor.

5. The headache of posterior fossa tumor is almost always over the back of the head, although it may occur elsewhere as well.

6. Headache is usually the first symptom of posterior fossa tumor except with cerebellopontile angle tumors.

7. Headache is the first symptom of one-third of supratentorial tumors.

8. The headache of cerebellopontile angle tumors is frequently and sometimes solely postauricular.

9. Headache from supratentorial tumors is rarely in the back of the head unless associated with papilledema.

10. When supratentorial tumors cause headache in the back of the head, headache in the front of the head is usually also present.

11. When headache is both frontal and occipital it indicates extensive displacement of the brain and has little localizing value.

12. Brain tumor headache is commonly intermittent but when it is continuous its value in localization is greatly enhanced.

Conclusions

1. Brain tumor headache is produced by traction upon intracranial pain sensitive structures, chiefly the large arteries, veins and venous sinuses, and certain cranial nerves. There are two types of traction which operate singly or in combination: local traction by the tumor mass upon adjacent structures; and distant traction by extensive displacement of the brain, either directly by the tumor or indirectly by ventricular obstruction (internal hydrocephalus).

2. As an aid in the localization of brain tumor, the value of headache is limited by two facts: the headache may be remote from the site of its production, and the site of production of the headache may be remote from the tumor.

3. In spite of these limitations, when it is interpreted in terms of known principles of intracranial pain production and pain reference, the headache of brain tumor may aid significantly in the diagnosis and localization of the lesion.

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PROCEEDINGS OF ACADEMY MEETINGS

STATED MEETINGS

FEBRUARY 5—*The New York Academy of Medicine.* Executive session — Reading of the minutes. ¶ Papers of the evening —*Scientific program in conjunction with the New York Tuberculosis and Health Association* — Recent developments in the treatment of pulmonary tuberculosis —a] Physiological considerations, Dickinson W. Richards, Jr., Associate Professor of Clinical Medicine, College of Physicians and Surgeons, Columbia University; b] Surgical treatment, Frank B. Berry, Assistant Clinical Professor of Surgery, College of Physicians and Surgeons, Columbia University. ¶ Report on election of Fellows. ¶ Report on election of trustee.

FEBRUARY 19—*The Harvey Society (in affiliation with The New York Academy of Medicine).* The Fifth Harvey Lecture, "Marijuana," Roger Adams, Professor of Chemistry, University of Illinois.

MARCH 5—*The New York Academy of Medicine.* Executive session—a] Reading of the minutes; b] Report of Nominating Committee. ¶ Papers of the evening—Headaches—a] General considerations, Harold G. Wolff, Associate Professor of Medicine, Cornell University Medical College; b] Clinical varieties and their treatment, Bayard T. Horton, Associate Professor of Medicine, Mayo Foundation. ¶ Report on election of Fellows, Honorary Fellow and Corresponding Fellow.

MARCH 19—*The Harvey Society (in affiliation with The New York Academy of Medicine).* The Sixth Harvey Lecture, "Physiologic Studies Pertaining to Aviation Medicine and Deep Sea Diving," Albert R. Behnke, Lieutenant Commander, Medical Corps, U. S. Navy, Instructor in charge of Department of Atmospheric Hygiene, Navy Yard.

SECTION MEETINGS

FEBRUARY 3—*Dermatology and Syphilology.* Presentation of cases—From the Diagnostic and Treatment Centers, Bureau of Social Hygiene of the New York City Department of Health; and Cooperative Clinics. ¶ Discussion. ¶ Executive session.

FEBRUARY 6—*Surgery.* Reading of the minutes. ¶ Papers of the evening—a] The method of measuring the electrical potential difference across the gastric membranes and its clinical significance (lantern slides), Edmund N. Goodman (by invitation); b] The circulation of the spleen in relation to spherocytic jaundice (lantern slides), Allen O. Whipple; c] Cardio-pulmonary functional tests in surgery, Herbert C. Maier, André Cournand (by invitation). ¶ General discussion. ¶ Executive session.

FEBRUARY 10—*Neurology and Psychiatry.* Reading of the minutes. ¶ Presentation of cases—From the service of The Mt. Sinai Hospital, Israel S. Wechsler; Discussion, Sidney Gross. ¶Papers of the evening—a] The aims and limitations of aptitude testing, Samuel P. Horton, Ph.D. (by invitation); Discussion by Gladys Tallman, Ph.D., Robert B. McGraw; b] Classification, diagnosis and treatment of spinal arachnoiditis, J. Lawrence Pool (by invitation); Discussion by Lewis D. Stevenson, Clement B. Masson, Charles Davison. ¶ Executive session.

FEBRUARY 10—*Joint Scientific Session Section of Pediatrics and the New York Heart Association* (Heart Committee of the New York Tuberculosis and Health Association. Papers of the evening—a] Prevention of rheumatic recurrences: a discussion of various measures now being used, Ann G. Kuttner (by invitation), Medical Director, Irvington House; b] Twenty-years observation of

children with rheumatic heart disease following convalescent care, Alexander T. Martin; c] Coagency in the approach to the problem of rheumatic heart disease, Edward S. Rogers (by invitation), Assistant Commissioner, Department of Health, State of New York. ¶ Discussion, Homer F. Swift, Katherine G. Dodge.

FEBRUARY 16 — *Ophthalmology.* Instruction Hour: 7:00 to 8:00 o'clock — Conjunctivitis, Alson Braley (by invitation). ¶ Executive session — Reading of the minutes. ¶ Presentation of cases — a] Recession of the trochlea for cyclotropia (two cases), Donald W. Bogart; b] A case of a tumor of the iris root, E. M. Katzin; c] A case of lacerated lid with coloboma—plastic repair, Rudolf Aebli. ¶ Papers of the evening—a] The relation of field contraction to blood pressure in cases of chronic primary glaucoma, Algernon B. Reese, John S. McGavic (by invitation); b] Virus diseases in ophthalmology. Phillips Thygeson; Discussion, I. Givner.

FEBRUARY 17—*Joint Meeting Section of Medicine and the New York Society of Tropical Medicine.* Papers of the evening—a] Florence Nightingale and tropical and military medicine, Thomas T. Mackie, Visiting Physician, Roosevelt Hospital; b]Tropical medicine in United States military history, Henry E. Meleny, Hermann M. Biggs Professor of Preventive Medicine, New York University; c] Typhus fever, John S. Snyder, Rockefeller Institute. ¶ Executive session of the section.

FEBRUARY 18 — *Genito-Urinary Surgery.* Reading of the minutes. ¶ Papers of the evening—a] Certain aspects of perinephritic infections (lantern slides), A. Raymond Stevens; b] Aneurysm of renal artery, C. G. Child (by invitation); c] Secondary hyperplasia of a parathyroid in renal insufficiency (report of a case), Allister McLellan, Irvin Balensweig; d] A possible relationship of lymphogranuloma and cystitis, Victor F. Marshall (by invitation); e] Occurrence and treatment of neurogenic bladder following resection of the rectum, Harmon J. Truax (by invitation); f] The treatment of prostatic cancers with endocrine therapy, Archie Dean; g] Relief of bladder neck and prostatic obstruction by transurethral resection, Frederick McLellan (by invitation); h] Causes of unsatisfactory end-results following adequate prostatectomy, Gustavus A. Humphreys (by invitation); i] Movie films demonstrating the physiology of spermatozoa, Robert Hotchkiss (by invitation). ¶ Discussion.

FEBRUARY 18—*Otolaryngology.* Reading of the minutes. ¶ Paper of the evening—Recent developments in chemotherapy, William D. Province (by invitation); Discussion by James G. Dwyer, J. Winston Fowler, Mervin C. Myerson, Arthur Palmer. ¶ General discussion. ¶ Executive session.

FEBRUARY 20—*Orthopedic Surgery.* Reading of the minutes. ¶ Presentation of case report—A case of static progressive spondylolisthesis, Nicholas S. Ransohoff. ¶ Papers of the evening—a] Double clothes-pin graft of the spine in spondylolisthesis and where laminar defects exist, David M. Bosworth; b] Traumatic spondylolisthesis, a report of three unusual cases in adults, Samuel Kleinberg, Michael S. Burman (by invitation). ¶ Discussion, Mather Cleveland. ¶ General discussion. ¶ Executive session. ¶ Honor guest and speaker, Dr. Luthero Vargas, Brazilian orthopedist and son of the President of Brazil, addressed the Section on "Centro Medico Pedigogico, Oswaldo Cruz, of Rio de Janeiro."

FEBRUARY 24—*Obstetrics and Gynecology*—Program presented by Sloane Hospital for Women. Executive session—Reading of the minutes. ¶ Papers of the evening—a] Local application of estrogens in treatment of pruritus vulvae, Charles Lee Buxton (by invitation); b] Use of Miller-Abbot tube in obstetrics and gynecology, W. E. Pollard

(by invitation); c] Anatomical changes following radiation for carcinoma of the corpus uteri, James A. Corscaden; d] Mechanisms for face and brow presentations, D. Anthony D'Esopo (by invitation). ¶ General discussion.

MARCH 3—*Dermatology and Syphilology.* Presentation of cases—a] City Hospital; b] New York Polyclinic Medical School and Hospital; c] Metropolitan Hospital. ¶ Discussion. ¶ Executive session, appointment of Nominating Committee.

MARCH 6—*Combined Meeting: Medicine and Surgery.* Reading of the minutes. ¶ Papers of the evening—a] The treatment of chronic arthritis with special reference to the use of gold salts in rheumatoid arthritis, Martin Henry Dawson; b] Surgical procedures found useful in treatment of rheumatoid arthritis (motion pictures), M. N. Smith-Petersen (by invitation); c] The treatment of Marie-Strumpell arthritis of the spine, William Sheppard (by invitation). ¶ General discussion. ¶ Executive session, appointment of Nominating Committee for each Section.

MARCH 10—*Combined Meeting: Neurology and Psychiatry and the New York Neurological Society.* ¶ Presentation of case—Paraphenylendiamine poisoning and the central nervous system, Charles Davison; Discussion by Moses Keschner. ¶ Papers of the evening—a] The development of neurological surgery in New York City during the past twenty-five years—with remarks on the advances due to experiences in the first World War (illustrated by lantern slides), Charles A. Elsberg; b] Neuropsychiatric problems in burn encephalopathies of childhood, Lauretta Bender; Discussion by Morris B. Bender. ¶ Executive session of the Section—a] Reading of the minutes; b] Appointment of Nominating Committee.

MARCH 11—*Historical and Cultural Medicine.* ¶ Executive session—Nomination of Section Officers and one member of Advisory Committee. ¶ Papers of the evening—a] The early history of auscultation and the stethoscope, Walter B. Mount; b] Selections from the Wolfe Collection of illustrations of noted men, Wardner D. Ayer (by invitation). ¶ Discussion.

MARCH 12—*Pediatrics.* ¶ Executive session—a] Reading of the minutes; b] Appointment of Nominating Committee. ¶ Papers of the evening—a] The management of acute obstructive laryngotracheobronchitis from the pediatric standpoint, A. Harry Neffson (by invitation); b] The treatment of pertussis, Lewis Jacobs (by invitation); c] The early management of poliomyelitis with an evaluation of the Sister Kenny Treatment, Mary M. I. Daly (by invitation), Jerome Greenbaum (by invitation), Edward Reilly (by invitation), Alvah Weiss (by invitation).

MARCH 16 — *Ophthalmology.* ¶ Instruction Hour: 7:00 to 8:00 o'clock; The use of the Cross Cylinder, Willis Knighton. ¶ Exhibit—Types of strabismus, James W. White, Harold W. Brown. ¶ Executive session—a] Reading of the minutes; b] Appointment of Nominating Committee. ¶ In memoriam—Julius Wolff, Charles H. May. ¶ Presentation of cases—a] A case of hypertelorism, Henry O. Little (by invitation); b] A case of Coates' disease, A. L. Kornzweig (by invitation); c] A case of nevus verrucosus of the scalp with involvement of the conjunctiva, A. Russell Sherman; Discussion by Milton L. Berliner. ¶ Papers of the evening—a] Multiple sclerosis as an etiological factor in retrobulbar neuritis, William L. Benedict, Rochester, Minnesota (by invitation); Discussion by Tracy Putnam; b] Eye dominance, its nature and treatment, Ira Wile.

MARCH 18—*Genito-Urinary Surgery.* ¶ Executive session—a] Reading of the minutes; b] Appointment of Nominating Committee. ¶ Papers of the evening—

a] An unusual case of renal carbuncle in a child, Joseph Tenenbaum; b] Primary carcinoma of the male urethra, Stanley Woodruff; c] Anuria and stone formation following sulfadiazine therapy, Howard Jeck, L. A. Orkin (by invitation); d] Evaluation of renal function tests in surgery, Mark Fishberg (by invitation); Discussion by Henry Bugbee and Abraham Hyman.

MARCH 18—*Otolaryngology (in joint session with the Section on Otolaryngology of the College of Physicians of Philadelphia)* Reading of the minutes. ¶ Papers of the evening—a] The specialist in the Army, Lieutenant Alfred W. Doust, M.C., U.S.A. (by invitation); b] Otolaryngological aspects of aviation, Lieutenant Commander Leon D. Carson, M.C., U.S.N. (by invitation); c] War casualties in the Navy, Captain Reynolds Hayden, M.C., U.S.N., former commanding Officer, Naval Hospital, Pearl Harbor (by invitation). ¶ General discussion. ¶ Executive session of the Academy Section, Appointment of Nominating Committee.

MARCH 20—*Orthopedic Surgery.* Reading of the minutes. ¶ Presentation of Apparatus—Demonstration of "hand holder," Edgar D. Oppenheimer. ¶ Papers of the evening—a] Open reduction and implantation of bone chips for ununited fracture of the femoral neck (5 cases), Halford Hallock; b] Ununited fractures, Fred H. Albee; Discussion by Lewis Clark Wagner. ¶ General discussion. ¶ Executive session, appointment of Nominating Committee.

MARCH 24—*Obstetrics and Gynecology.* ¶ Executive session—a] Reading of the minutes; b] Appointment of Nominating Committee. ¶ Presentation of Cases* —a] Chronic inversion of the uterus; b] Ovarian pregnancy, William E. Alsop (by invitation). ¶ Papers of the evening—a] End-results in cancer of the ovary*, H. C. Taylor, Jr., A. V. Greeley; b] A follow-up evaluation of the Richardson Composite Operation for uterine prolapse, Richard W. TeLinde, Chief Gynecologist, Johns Hopkins Hospital (by invitation); Discussion by Albert H. Aldridge. ¶ General discussion.

*Case reports and paper (a) presented by members of Gynecological Staff of Roosevelt Hospital.

AFFILIATED SOCIETIES

FEBRUARY 16—*New York Roentgen Society (in affiliation with The New York Academy of Medicine).* Papers of the evening —a] The role of the roentgenologist in orthopedic surgery, Samuel Kleinberg; b] X-ray diagnosis in early epiphyseolysis, Leo Mayer; c] Roentgenographic aids in the treatment of scoliosis, Samuel A. Jahss (by invitation); d] Skeletal changes in chronic renal insufficiency, Maurice M. Pomeranz. ¶ General discussion. ¶ Executive session.

FEBRUARY 26—*New York Pathological Society (in affiliation with The New York Academy of Medicine).* Presentation of cases—a] Syphilis of the aorta and coronaries, George Strassman, Philip Goldstein (by invitation); b] Squamous cell carcinoma of the bronchus and spindle cell sarcoma of the lung, Daniel A. Rock (by invitation), John W. Hall (by invitation). ¶ Papers of the evening—a] Liver lesions caused by excess ingestion of sulphur-containing amino acids, Joseph Victor (by invitation), David P. Earle, Jr. (by invitation); b] Agnogenic myeloid metaplasia of the spleen, Carl Reich, James Lisa. ¶ Executive session.

AFFILIATED SOCIETIES

MARCH 16—*New York Roentgen Society (in affiliation with The New York Academy of Medicine).* Paper of the evening—Certain diagnostic problems in the roentgenological examination of the esophagus, Richard Schatzki (by invitation). ¶ General discussion opened by Ross Golden. ¶ Executive session.

MARCH 26—*New York Pathological Society (in affiliation with The New York Academy of Medicine).* ¶ Papers of the evening—a] Benign chondroblastoma of bone: remarks on the so-called calcifying or chondromatous giant cell tumor, Louis Lichtenstein, Henry L. Jaffe; b] Benign and malignant mammary alterations produced in the rat by methylcholanthrene and crystalline estrogen, Milton J. Eisen; c] Spontaneous solitary and multiple mast cell tumors (mastocytomas) in dogs, Frank Bloom. ¶ Executive session.

DEATHS OF FELLOWS

FINKLE, PHILIP: 1000 Park Avenue, New York City; born in Hartford, Connecticut, November 2, 1894; died in Miami Beach, Florida, March 13, 1942; graduated in medicine from the College of Physicians and Surgeons, Columbia University, in 1918; elected a Fellow of the Academy May 7, 1931.

Dr. Finkle was adjunct physician and physician-in-charge of the arthritic clinic at The Mount Sinai Hospital and was engaged in medical research. He was a Fellow of the American College of Physicians and a member of the State and County Medical Societies.

FISCHER, CARL FERDINAND HERMANN: 35 East 84 Street, New York City; born in Clausthal, Province of Hannover, Germany, April 24, 1871; died in New York City, March 5, 1942; graduated in medicine from the College of Physicians and Surgeons, Columbia University, in 1896; elected a Fellow of the Academy February 6, 1908.

Dr. Fischer joined the Lenox Hill Hospital staff in 1896 and for the past six years served in the capacity of consulting surgeon and head of the department of medical education. He was professor of clinical surgery at New York University College of Medicine, 1931-36; lecturer on clinical surgery at the College of Physicians and Surgeons, Columbia University, 1911-15; lecturer on clinical pathology at Polyclinic Medical School and Hospital, 1900-03; and for a number of years was consulting surgeon to the Rockaway Beach Hospital. He was a Fellow of the American College of Surgeons, a member of the American Association for Thoracic Surgery, the New York Surgical Society, and the State and County Medical Societies.

GARVIN, WILLIAM CREIGHTON: Binghamton, New York; born in Philadelphia, Pennsylvania, November 30, 1873; died in Binghamton, April 3, 1942; graduated in medicine from the College of Physicians and Surgeons, Columbia University, in 1903; elected a Fellow of the Academy November 2, 1922.

Dr. Garvin was superintendent of the Binghamton State Hospital, a Fellow of the American Medical Association, a member of the American Psychiatric Association, a member of the American Psychopathological Association, and a member of the State and County Medical Societies.

GREENWALD, HARRY MORDECAI: 499 Ocean Avenue, Brooklyn, New York; born in Unguar, Hungary, March 18, 1890; died in Brooklyn, March 15, 1942; graduated in medicine from Bellevue Medical School, New York University (now New York University College of Medicine) in 1910; elected a Fellow of the Academy April 6, 1933.

Dr. Greenwald was attending pediatrician to the Israel Zion Hospital and chief pediatrician to the Trinity Hospital. He was a diplomate of the American Board of Pediatrics, a Fellow of the American Medical Association, a member of the American Academy of Pediatrics, and a member of the State and County Medical Societies.

HIRSCH, ISAAC SETH: 133 East 58 Street, New York City; born in New York City,

December 3, 1880; died in New York City, March 24, 1942; graduated in medicine from the College of Physicians and Surgeons, Columbia University, in 1902; elected a Fellow of the Academy February 6, 1913.

Dr. Hirsch was professor of radiology at New York University College of Medicine; x-ray consultant to the New York State Compensation Commission; and director of radiology at New York University Clinic and Beth Israel Hospital. From 1914 to 1917, he was professor of roentgenology at New York Post-Graduate Medical School and Hospital; and from 1910 to 1926, he was director of the department of radiology at Bellevue Hospital. In 1920, Cambridge University (England) awarded him a diploma in radiology. He was a diplomate of the American Board of Radiology, a Fellow of the American College of Physicians, a Fellow of the American Medical Association, a member of the Radiological Society of North America, Inc., and its vice-president in 1927-28, and a member of the State and County Medical Societies.

Dr. Hirsch was the author of "Principles and Practice of Roentgenological Technique," 1920; and "Principles and Practice of Roentgen Therapy," 1923. Besides his numerous writings, he served as associate editor and director of the department of technique and new devices of the American Journal of Electrology and Radiology.

In 1917 he entered the Medical Corps of the United States Army as Captain and later was promoted to the rank of Major.

JOHNSON, FREDERIC MORTIMER, JR.: 25 Morris Street, Yonkers, New York; born in New York City, August 5, 1874; died in Yonkers, New York, April 13, 1942; graduated in medicine from Syracuse University College of Medicine in 1904; elected a Fellow of the Academy February 3, 1910.

Dr. Johnson was attending gastroenterologist to the St. John's Hospital at Riverside, a diplomate of the American Board of Internal Medicine, a Fellow of the American Medical Association, a member of the National Gastroenterological Association, and a member of the State and County Medical Societies.

JUDD, ASPINWALL: 50 East 75 Street, New York City; born in St. Louis, Missouri, in 1868; died in New York City, March 17, 1942; graduated in medicine from the College of Physicians and Surgeons, Columbia University, in 1896; elected a Fellow of the Academy May 19, 1904. He was a Fellow of the American Medical Association and a member of the State and County Medical Societies.

MCNEILL, WALTER HAROLD, JR.: 115 East 61 Street, New York City; born in Brooklyn, New York, November 13, 1886; died in New Rochelle, New York, March 22, 1942; graduated in medicine from Cornell University Medical College in 1910; elected a Fellow of the Academy February 1, 1917.

Dr. McNeill was clinical professor of the department of urology at New York University College of Medicine; attending urologist to Bellevue Hospital, the Welfare Hospital for Chronic Diseases, the Mount Vernon Hospital and the New Rochelle Hospital; and consulting urologist to the French Hospital and New York Central Railroad. He was a diplomate of the American Board of Urology, a Fellow of the American College of Surgeons, a Fellow of the American Medical Association, and a member of the American Urological Association, and the State and County Medical Societies.

MOORE, ALBERTUS ADAIR: 140 East 54 Street, New York City; born in Camden, South Carolina, June 26, 1876; died in New York City, March 23, 1942; graduate in medicine from the Medical College of the State of South Carolina in 1896; elected a Fellow of the Academy April 1, 1909. He was a Fellow of the American Medical Association and a member of the State and County Medical Societies.

ROBINSON, DAISY MAUD ORLEMAN: Meridale, New York; born in Fort Riley, Kansas, November 6, 1869; died in Jacksonville, Florida, March 12, 1942; graduated in medicine from the Medical Department, Columbian Medical College (now George Washington University School of Medicine), Washington, D. C., in 1890; elected a Fel-

low of the Academy, May 6, 1897.

Dr. Robinson was associated with the New York State Department of Health and with the U. S. Public Health Service in Washington. She was a member of the Société française de dermatologie et de syphiligraphie, being the first woman elected to membership in this Society, and a member of the State and County Medical Societies.

SKEEL, HARRY ROBERTSON: 115 East 61 Street, New York City; born in New York City, October 4, 1885; died in Riverdale, New York, March 22, 1942; graduated in medicine from the College of Physicians and Surgeons, Columbia University, in 1913; elected a Fellow of the Academy April 6, 1933.

Dr. Skeel was surgical director of ophthalmology at the Manhattan Eye, Ear and Throat Hospital, and consulting ophthalmologist to the Bronx Eye and Ear Hospital. He was a Fellow of the American Medical Association, a member of the Association for Research in Ophthalmology, Inc., and a member of the State and County Medical Societies.

STEARNS, HENRY SCHERMERHORN: 655 Park Avenue, New York City; born in Yonkers, New York, December 4, 1861; died in New York City, April 2, 1942; graduated in medicine from the Medical Department of the University of the City of New York in 1884; elected a Fellow of the Academy June 6, 1889. He was a member of the New York County Medical Society, and its president, 1904-1905, a member of the State Medical Society, and a Fellow of the American Medical Association.

WALKER, JOHN BALDWIN: 117 East 72 Street, New York City; born in Lodi, New Jersey, January 16, 1860; died in New York City, April 13, 1942; received the degree of B.A. from Harvard University in 1884; graduated in medicine from Harvard Medical School in 1888; elected a Fellow of the Academy March 2, 1893; and served the Section of Surgery as its Secretary from 1896 to 1901 and as its Chairman from 1901 to 1903.

After three years of postgraduate work in Europe, Dr. Walker joined the staff of the New York Polyclinic Medical School and Hospital in 1894, and from 1897 to 1910 he served this institution as instructor in operative surgery. He was professor of clinical surgery at the College of Physicians and Surgeons, 1910-1938; and for many years consulting surgeon to Bellevue and Manhattan State Hospitals and the Hospital for Special Surgery, formerly the Hospital for the Ruptured and Crippled. He was a Fellow of the American College of Surgeons, and a member of the American Medical Association, the American Surgical Association, the State and County Medical Societies and a member of the Société internationale de chirurgie.

Dr. Walker was the author of "Volume IX—Hernia" in "Twentieth Century Practice of Medicine," and contributed many papers on the operative treatment of fractures.

During the last war, Dr. Walker commanded Base Hospital 116 of the American Expeditionary Force in France and for his work was awarded the Distinguished Service Medal. He held the rank of Colonel in the Officers Reserve Corps.